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ON EPILEPSY, PAIN, PARALYSIS, ETC.

LECTURES

ON

EPILEPSY, PAIN, PARALYSIS,

AND CERTAIN OTHER

DISORDERS OF THE NERVOUS SYSTEM.

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BY

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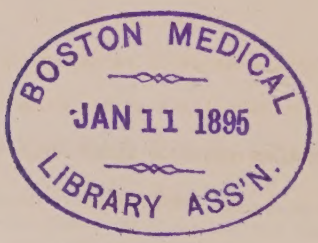

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Dedicated

AS A

TRIBUTE OF HIGH ESTEEM AND ADMIRATION

TO

THOMAS WATSON, M.D. CANTAB.,

D.C.L OXON., F.R.S.,

PRESIDENT OF

THE ROYAL COLLEGE OF PHYSICIANS OF LONDON,

PHYSICIAN EXTRAORDINARY TO HER MAJESTY THE QUEEN,

CONSULTING PHYSICIAN TO KING'S COLLEGE HOSPITAL,

ETC. ETC.

PREFACE.

Upwards of twelve years ago I published a small work in which my principal object was to show that it was necessary to revise the theory of muscular motion and to consider muscular contraction as a physical process in which the attractive force inherent in the physical constitution of the muscular molecules was the contractile agent: and on more than one occasion subsequently I have again ventured into print with a view to enforce the same object. Concerning the first of these attempts* I may say what Dryden said concerning one of his own plays,—"it was only a confused mass of thoughts tumbling over one another in the dark, when the fancy was yet in its first work, moving the sleeping images of things toward the light, there to be distinguished, and then either chosen or rejected

* "Philosophy of Vital Motion." 8vo. London: Churchill, 1851.

by the judgment." And I look back with some dissatisfaction even at the last of these attempts,* not because my convictions have for a moment wavered as to the truth of the principle for which I am doing battle, but because I find that I can now support these convictions by much additional evidence—by evidence which is indispensable as well as new.

In the present course of lectures I have more to do than I may hope to do well in the time allotted to me. I have again to show that a radical change is necessary in the current physiology of muscular motion and in the current pathology and therapeutics of convulsion, tremor, and spasm; and in doing this, I have much to say that is altogether new, and something to omit of what I have said previously. In my remarks upon the physiology of muscular motion, for example, I have to begin from a new starting-point. I have to give up regarding the "muscular current," and the "nerve current," upon which I have said so much as primary phenomena in animal electricity. I have to show that

* "Epileptic and other Convulsive Affections of the Nervous System, their Pathology and Treatment." Third Edition (incorporating the Gulstonian Lectures for 1860). Post 8vo. London: Churchill, 1861.

the natural electricity of muscle and nerve during the state of rest is in a statical, and not in a current condition—a condition which is no other than that of tension. I have to show that the electrical discharge, analogous to that of the torpedo, which M. Matteucci has shown to accompany muscular action, is essential to the interpretation of muscular action. I have to show, in short, that it is necessary to borrow light from the discoveries of M. Matteucci in natural electricity as well as from those of M. Du Bois-Reymond in the same department of science, in order to obtain a clear insight into the physiology of muscular motion and into the pathology and therapeutics of convulsion, tremor, and spasm; and that, with this light, it is possible to find the truth by a much easier and shorter way, and to apprehend it with far greater distinctness. I have also to take a step in advance of my old position and to show that the same radical change which is necessary in the physiology of muscular motion and in the pathology and therapeutics of convulsion, tremor, and spasm, is also necessary in the physiology of sensation and in the pathology and therapeutics of pain. And, lastly, I have to point out certain

changes in the theory and treatment of paralysis which appear to be necessitated by the physiological, pathological, and therapeutical premises.

25 Cavendish Square.

CONTENTS.

LECTURE I.

INTRODUCTORY REMARKS.—Object in view—plan of lectures.

PHYSIOLOGY OF ORDINARY MUSCULAR MOTION.—*Preliminary Matters.*—Animal electricity supplying master-key—Humboldt's estimate of the importance of animal electricity in physiology—Historical particulars respecting animal electricity—Galvani—Volta—Alexander von Humboldt—Aldini—Nobili—Matteucci—Du Bois-Reymond.

The electrical phenomena presented by living muscle and nerve during the state of rest.—The longitudinal and transverse surfaces of the fibers in a state of electric antagonism, the former surfaces being electrified positively, the latter surfaces negatively. Electric tension different at different points of the longitudinal and transverse surfaces—the ordinary electric antagonism of the longitudinal and transverse surfaces reversed under certain circumstances—galvanometric signs of animal electricity—electroscopic signs of animal electricity—all signs of animal electricity absent in rigor mortis—primary electrical condition of living muscle and nerve during rest that of statical electricity—the "muscular current," and the "nerve current" only secondary phenomena—the presence of the natural electricity of the muscle may give rise to the state of muscular relaxation by keeping the muscular molecules in a state of electrical tension—the absence of this electricity may bring about rigor mortis by allowing the physical attraction of the muscular molecules to come into play. Pp. 25–53.

LECTURE II.

The electrical phenomena presented by muscle and motor nerve during the state of action.—The state of action in muscle is accompanied by a discharge of electricity analogous to that of a torpedo—the state of

action in a motor nerve is accompanied by a discharge of electricity analogous to that of a torpedo—the natural electricity which is present in living muscle during the state of rest is almost altogether absent during the state of action—the natural electricity which is present in motor nerve during the state of rest is almost altogether absent during the state of action—ordinary muscular contraction and rigor mortis may alike be dependent upon the absence of the natural electricity which is present in living muscle during the state of rest and relaxation.

The part which animal electricity has to play in the process of muscular motion.—Instantaneous currents of high-tension electricity, as the currents of a coil-machine, have the power of producing a state of action in motor nerve and muscle—continuous currents of low-tension electricity, such as the common galvanic current, have a paralyzing influence upon motor nerve and muscle—a motor nerve or muscle cannot be thrown into a state of action by artificial electricity unless it retain a certain amount of natural electricity—natural electricity is diminished or discharged when a motor nerve is thrown into a state of action by artificial electricity—difference of the action of the two galvanic poles upon the natural electricity of nerve, the positive pole increasing, the negative pole decreasing, this electricity—augmented irritability associated with diminished natural electricity—irritability lost when electricity is extinguished—augmentation of electric tension in natural electricity involves suspension of irritability—influence of positive galvanic pole is to maintain and restore natural electricity—influence of negative galvanic pole is to extinguish natural electricity and to destroy (except for a moment or two) irritability—explanation of "voltaic alternatives"—explanation of the action of a centrifugal or "direct" galvanic current upon a motor nerve—explanation of the action of a centripetal or "inverse" galvanic current upon a motor nerve—differences in the action of the "direct" and "inverse" galvanic current upon a nerve to be ascribed to the negative pole being next the muscle in the former case and to the positive pole being in this position in the latter case, and not to differences in the direction of the current—artificial electricity acts upon muscle and upon nerve in the same manner—the results of exposing motor nerve or muscle to the action of artificial electricity appear to depend altogether upon the necessary reactions which take place between artificial and natural electricity. Pp. 54–82.

LECTURE III.

The part which the blood has to play in the process of muscular motion.—Rigor mortis is associated with stagnation and coagulation of the blood—muscles which have passed into the state of rigor mortis will return into the state of vital relaxation if their vessels be again supplied with blood—the action of the blood produces vital relaxation in muscle—rigor mortis associated with absence of this action—increased disposition to ordinary muscular contraction the result of a diminished supply of blood to the muscular system—general convulsion brought about by sudden hæmorrhage—general convulsion brought about by sudden suffocation—the action of strychnia and brucia in producing spasm equivalent, in a certain sense, to loss of arterial blood—influence of venous blood in the process of muscular action equivalent to the absence of the influence of arterial blood, and to no more than this—as in rigor mortis, so in ordinary muscular contraction, the contracted condition of the muscle is associated with the absence of that action of the blood which seems to have to do with the production of vital relaxation in living muscle.

The part which "nervous influence" has to play in the process of muscular motion.—Rigor mortis is associated with entire absence of "nervous influence"—ordinary muscular contraction is associated with deprivation of "nervous influence," and not with a contrary state of things—muscles do not pass into a state of contraction when they may be supposed to receive a larger supply of "nervous influence" than usual—power of muscular contraction appears to be inversely related to the amount of "nervous influence" supplied to the muscles from the great nerve-centers—"augmented irritability" does not imply augmented vitality in nerve or muscle—difficult to believe that "nervous influence" produces muscular contraction by playing the part of a stimulus to a vital property of contractility in muscle—not difficult to believe that "nervous influence" may play its part in the process of muscular motion through the instrumentality of the natural electricity of the nervous system—nervous action may be the result of a state of reversal in the electrical relations of the exterior and interior of the nerve-fibers in some parts of the nervous system—such reversal will give rise to an electrical discharge (analogous to that of the torpedo) in the neighborhood of the nerve, and this discharge produces muscular contraction by suspending for the moment the natural electricity of the muscle—hæmorrhage and suffocation may bring about

convulsion by producing that reversal in the electrical relations of the exterior and interior of the nerve-fibers in some parts of the nervous system which brings about nervous action—nervous action arising from local causes, mechanical or other, may be explained in the same way—the state of nerve known as "irritation" the natural result of the electrical reversal of which mention is here made—the will may avail itself of the aid of the natural electricity of the nervous system in producing voluntary muscular action—"nervous influence," apart from nerve-electricity, is, to say the least, a very indefinite idea.

The part which certain comparatively unimportant non-electrical agents have to play in the process of muscular motion.—All these agents produce a reversal in the electrical relations of the exterior and interior of the nerve in the part acted upon, and the state of action which results is the consequence of this reversal, as before explained. Pp. 83–121.

LECTURE IV.

The theory of muscular motion which appears to arise out of the premises.—This theory appears to be a purely physical theory—general features of this theory as deduced from evidence advanced hitherto—this theory not contradicted by evidence still to be advanced—natural electricity of muscle not converted into the contractile force of muscle—law of muscular contraction a physical law—the fact that dead muscle is less tough and strong than living muscle admits of a physical explanation—the fact that muscle cannot act without intervals of rest admits of a physical explanation—the fact that muscle may contract and relax without change of volume and without loss of time has its strict parallel among purely physical phenomena—increased disposition to muscular contraction not connected with a state of exalted functional activity in a vital property of irritability in motor nerve or muscle—this theory of muscular motion is confirmed in no small degree by explaining muscular relaxation no less than muscular contraction, by being equally applicable to ordinary muscular contraction and to rigor mortis, and by bringing the phenomena of muscular motion into subjection to physical law.

Physiology of Rhythmical Motion.—*The beating of the heart.*—The blood plays the same part in the movements of the ventricles and in ordinary muscular motion—the same remark applies to "nervous influence"—the differences which distinguish the rhythm of the auri-

cles from that of the ventricles may be resolved, in the main, into simple consequences of the movements of the ventricles—the manner in which the movements of the heart are affected by electrifying the medulla oblongata or pneumogastric nerve is sufficiently intelligible—the beating of the heart, or of certain fragments of a heart, after removal from the body, not altogether unintelligible—the action of the vaso-motor nerves upon the vessels in harmony with the conclusion respecting the action of the heart and of the muscular system generally—intelligible explanation of "capillary force" a collateral result—intelligible explanation of the action of the heart a direct result of the previous reasonings respecting muscular motion.

The peristaltic movements of the alimentary canal.—The blood acts in these movements as it acts in the movement of the heart and in muscular motion generally—the way in which these movements are affected by electrifying the spinal cord or grand sympathetic in harmony with what has gone before—the movements in question are not unintelligible when regarded from the previous point of view.

The respiratory movements of the chest.—Nothing peculiar in the manner in which these movements are affected by "nervous influence"—these movements become more intelligible when interspersed in accordance with the view of muscular motion set forth in these lectures.

Conclusion.—This theory of ordinary muscular motion is applicable to rhythmical muscular motion, and an insight into the cause of the rhythm a result of its application—the theory of muscular motion set forth in these lectures derives no small degree of confirmation in the fact that it leads up a step nearer to the discovery of a common law for organic and inorganic nature.

The Physiology of Sensation.—*The part which animal electricity has to play in the process of sensation.*—In the case of a sentient nerve, as in the case of a motor nerve, the nerve loses electricity when it passes from the state of rest into that of action.

The part which artificial electricity has to play in the process of sensation.—The change in a sensory nerve when sensation is produced by the action of voltaic electricity, and the change in a motor nerve when muscular contraction is produced by the same cause, are exact equivalents.

Conclusion respecting sensation.—There is no essential difference between the action which issues in sensation and the action which issues in muscular contraction.

General conclusion.—The problem of muscular motion and the problem of sensation are only to be solved when the agency of animal electricity is employed as the master-key. Pp. 122–157.

LECTURE V.

Convulsion.—*The Pathology of Convulsion.*—The epileptic and epileptiform paroxysm not unfrequently preceded by signs of defective respiration—this paroxysm usually accompanied by a state of unmistakable suffocation—the convulsion of hysteria or chorea usually associated with a state of very defective respiration—the condition of the respiration in convulsion supports the notion that convulsion is connected with depressed and not with exalted vitality. The interparoxysmal state in convulsive disorders is usually marked by evident signs of a feeble circulation—the epileptic and epileptiform paroxysm is usually if not invariably ushered in by signs of failure in the circulation—the strong, full, and frequent pulse of this paroxysm is the natural pulse of suffocation, the pulse of arteries carrying *black*, not red blood—convulsion is never coincident with an overactive state of the circulation—the convulsion which may attend upon the close of Bright's disease is connected with a pale and watery condition of the blood, and, it may be, with uræmia—epileptiform convulsion a direct consequence of sudden hæmorrhage—the condition of the circulation during convulsion is one which supports the notion that convulsion is connected with depressed, and not with exalted, vital power. Signs of wanting brain-power are scarcely ever absent in persons who are liable to convulsion in any form—all signs of mental life are abolished, or all but abolished, in the convulsive paroxysm—there is no clinical evidence to show that convulsion is associated with an overactive condition of the circulation in the brain—the appearances after death do not show that convulsion is necessarily connected with an inflammatory condition of any one of the great nerve-centers—convulsion must not be looked upon as a symptom of a congested condition of the cerebral veins—the peculiar condition of the nervous system known under the name of "irritation," and which in the majority of cases has a great deal to do with the production of convulsion, is in no sense the equivalent of inflammation—the phenomenon of aura or globus not at variance with the premises—nor yet the phenomena of "morbid irritability"—the condition of the respiration and

circulation in convulsion necessitates the conclusion that convulsion is connected with a state of depressed nervous energy, and not with a contrary state of things.

General conclusion respecting convulsion.—The condition of the respiration, circulation, and innervation during convulsion warrants the conclusion that this disorder is connected with depressed and not with exalted vital energy. Pp. 158–190.

LECTURE VI.

The Therapeutics of Convulsion.—The diet in many cases of convulsive disorder to contain somewhat more than an average quantity of oily and fatty matter and somewhat less than an average quantity of lean meat—suitable gymnastic exercises often very beneficial—purgatives to be avoided—bromide of potassium invaluable in many cases—cod-liver oil often very beneficial—phosphorus indicated not unfrequently—belladonna of questionable utility—opium a not unlikely remedy in certain cases and in certain contingencies—zinc of doubtful efficacy in ordinary epilepsy—cases in which zinc may be beneficial—alcoholic stimulants very trustworthy antispasmodics in the prevention and treatment of convulsion—blood-letting may be required to prevent certain consequences of convulsion—the therapeutics of convulsion to be based upon the notion that vital power has to be reinforced, and not upon the contrary notion. Pp. 191–215.

LECTURE VII.

Tremor.—*The Pathology of Tremor.*—The condition of the respiration in tremor warrants the belief that this disorder is connected with depressed and not with exalted vital power. The condition of the circulation during tremor is one of unmistakable depression—there is something uncongenial between tremor and an excited state of the circulation—the condition of the circulation in tremor warrants the belief that this disorder is connected with depressed and not with exalted vital energy. The condition of the brain during trembling one of unmistakable functional depression—something apparently uncongenial between tremor and an excited condition of brain—the state of the innervation generally during tremor warrants the belief that this

disorder is connected with depressed and not with exalted vital energy—cessation of tremor during sleep no objection to this view.

General conclusion respecting tremor.—The condition of the respiration, circulation, and innervation during tremor warrants the belief that this disorder is connected with depressed and not with exalted vital energy.

Therapeutics of tremor.—Means to be employed in the treatment of tremor those which exalt vital tone in general and nerve tone in particular.

Spasm.—*Pathology of spasm.*—Spasm associated with a depressed condition of the respiration. Spasm frequently associated with a depressed state of the circulation—spasm antagonized rather than favored by an excited state of the circulation. Spasm associated with deficient brain power—spasm antagonized rather than favored by inflammatory excitement in the nervous system—all nervous power at a very low ebb during spasm.

General conclusion respecting spasm.—The condition of the respiration, circulation, and innervation during spasm warrants the conclusion that this disorder is connected with depressed and not with exalted vital energy.

General conclusion respecting convulsion, tremor, and spasm.—Key to the pathology of spasm, tremor, and convulsion to be found in the view of the physiology of muscular motion set forth in these lectures, and this view of the physiology of muscular motion confirmed and established by the facts of pathology.

Therapeutics of spasm.—Means to be employed in the treatment of spasm those which are calculated to exalt vital energy in general, and nerve energy in particular. Pp. 216–240.

LECTURE VIII.

Pain.—*Pathology of pain.*—Neuralgic pain may be associated with a deeply depressed condition of the circulation—neuralgic pain antagonized rather than favored by an overactive condition of the circulation—pain the result of tenderness, and not pain of a neuralgic character, associated with a state of active congestion or inflammation. Condition of respiration during pain does not shed any very certain light upon the pathology of pain. Neuralgic pain antagonized rather than favored by inflammatory excitement of the nervous sys-

tem—pain the result of tenderness, not pain of a neuralgic character, associated with inflammatory excitement of the nervous system—neuralgic pain associated with a state of irritation in the nervous system, not with a state of inflammation—inflammation in the nervous system a consequence, not a cause, of the state of which neuralgic pain is the sign. Neuralgic pain, like convulsion, tremor, and spasm, to be regarded as a sign of defective vital power in general, and defective nerve power in particular—this view of the pathology of pain in perfect accordance with the view of sensation propounded in these lectures, and these two views reciprocally interpret and corroborate each other.

Therapeutics of pain.—Remarks confined to pain of a neuralgic character—avoidance of damp and cold of great importance—diet not to contain too much lean meat and too little fatty and oily matter—sugar in excess may be harmful—properly regulated use of alcoholic drinks an essential part of the preventive and curative plans of treatment—coffee, chocolate, or cocoa to be preferred as a common beverage to tea—habitual use of purgatives and aperients pernicious—cod-liver oil a very suitable tonic—phosphorus sometimes indicated—electricity in certain forms likely to be very useful—counter-irritants of much use—sedatives in sedative doses not required.

Paralysis in certain aspects.—*Certain questions relating to pathology.*—The fact that paralysis is accompanied by morbid muscular contraction, as twitchings, cramps, convulsions, and so on, no certain proof that the paralyzing lesion is of a congestive or inflammatory character, but rather an argument to the contrary—the fact that paralysis is accompanied by pain of a neuralgic character is no certain proof that the paralyzing lesion is of a congestive or inflammatory character, but rather an argument to the contrary—the fact that paralysis is accompanied by morbid sensations, such as formication, itching, pricking, coldness, heat, weight, tightness, and so on, is no certain proof that this paralyzing lesion is of a congestive or inflammatory character—the fact that paralysis is *not* accompanied by morbid muscular contractions, or by pain and other morbid sensations, is no certain proof that the paralyzing lesion is *not* of a congestive or inflammatory character—"late rigidity" has close analogies to rigor mortis—increased disposition to reflex movements no proof that the paralyzing lesion is of a congestive or inflammatory character—absence of paralysis in certain cases where integrity of nerve is destroyed in certain parts not altogether unintelligible.

Therapeutics of paralysis.—Preliminary remarks—proper use of electricity of much service in many cases—proper use of movements and manipulations of much service in many cases. Pp. 241–275.

Appendix.—Concerning some historical matters. Pp. 277–280.

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ON CERTAIN DISORDERS OF

THE NERVOUS SYSTEM,

WITH

SPECIAL REFERENCE TO A PROPOSED CHANGE IN THEORY AND TREATMENT.

LECTURE I.

In these Lectures the ultimate aim I have in view is to show that a fundamental change is necessary in the theory and treatment of all disorders of the nervous system which are characterized by convulsion or pain, or by any symptom analogous to convulsion or pain. With respect to muscular action, the current doctrine is—that muscle is endowed with a vital property of contractility,—that muscular contraction is the sign of vital excitement in this property,—that excessive muscular contraction, whether voluntary or involuntary, betokens excessive vital excitement in this property,—and that the treatment required in order to subdue convulsion, or any disorder analogous to convulsion, is one which is calculated to quieten excessive vital excitement. What I hope to do, is to show that the facts, old and new, but particularly those which have been brought to light during the last fifteen or twenty years, necessitate a very dif-

ferent doctrine, and a not less different practice. With respect to sensation, the current doctrine is, —that certain kinds of nerve-tissue are endowed with a vital property of sensibility,—that sensation is the sign of vital excitement in this property,— that pain, or any sensation analogous to pain denotes excessive vital excitement,—and that the treatment of pain and sensations analogous to pain must be ruled according to this view, the proper means being those which are calculated to subdue vital excitement. What I hope to do, is to show that the change in doctrine and practice which is demanded in the case of disordered muscular action is also demanded in the case of disordered sensation.

In carrying out this object, I shall speak in succession—

I. ON MUSCULAR MOTION.

A. ON SIMPLE MUSCULAR MOTION.

§ I. *On the part which animal electricity has to play in the process of muscular motion.*

(1) On some preliminary matters.
(2) On the electrical phenomena which belong to living muscle and motor nerve in the state of inaction.
(3) On the electrical phenomena which belong to muscle and motor nerve in the state of action.

§ II. *On the part which artificial electricity has to play in the process of muscular action.*

§ III. *On the part which certain non-electrical agents have to play in the process of muscular action.*

(1) On the part which the blood has to play in the process of muscular action.
(2) On the part which "nervous influence" has to play in the process of muscular motion.

(3) On the part which certain other non-electrical agents have to play in the process of muscular motion.

§ IV. *On the conclusions respecting muscular action which appear to arise out of the premises.*

B. ON RHYTHMICAL MUSCULAR MOTION.

1. *On the rhythmical movements of the heart.*
2. *On the peristaltic movements of the alimentary canal.*
3. *On the respiratory movements of the chest.*

II. ON SENSATION.

§ I. *On the part which animal electricity has to play in the process of sensation.*

§ II. *On the part which artificial electricity has to play in the process of sensation.*

§ III. *On the conclusions respecting sensation which appear to arise out of the premises.*

III. ON CONVULSION.

§ I. *On the pathology of convulsion.*

(1) On the pathology of convulsion as deduced from the condition of the respiration in convulsion.
(2) On the pathology of convulsion as deduced from the condition of the circulation in convulsion.
(3) On the pathology of convulsion as deduced from the condition of the innervation in convulsion.

§ II. *On the therapeutics of convulsion.*

IV. ON TREMOR.

§ I. *On the pathology of tremor.*

(1) On the pathology of tremor as deduced from the condition of the respiration in tremor.
(2) On the pathology of tremor as deduced from the condition of the circulation in tremor.

(3) On the pathology of tremor as deduced from the condition of the innervation in tremor.

§ II. *On the therapeutics of tremor.*

V. ON SPASM.

§ I. *On the pathology of spasm.*

(1) On the pathology of spasm as deduced from the condition of the respiration in spasm.
(2) On the pathology of spasm as deduced from the condition of the circulation in spasm.
(3) On the pathology of spasm as deduced from the condition of the innervation in spasm.

§ II. *On the therapeutics of spasm.*

VI. ON PAIN.

§ I. *On the pathology of pain.*

(1) On the pathology of pain as deduced from the condition of the respiration in pain.
(2) On the pathology of pain as deduced from the condition of the circulation in pain.
(3) On the pathology of pain as deduced from the condition of the innervation in pain.

§ II. *On the therapeutics of pain.*

VII. ON PARALYSIS IN CERTAIN ASPECTS.

§ I. *On certain questions relating to the pathology of paralysis and arising out of the previous inquiries.*

§ II. *On certain questions relating to the therapeutics of paralysis.*

Before proceeding to the pathological and therapeutical portions of my subject, I propose to speak at some length on the physiology of muscular action and sensation. I have, indeed, no other course open

to me: for I have to prepare the ground in physiology before I can hope to build securely in pathology and therapeutics.

I. ON MUSCULAR MOTION.

A. ON SIMPLE MUSCULAR MOTION.

§ I. ON THE PART WHICH ANIMAL ELECTRICITY HAS TO PLAY IN THE PROCESS OF MUSCULAR MOTION.

(1) *ON SOME PRELIMINARY MATTERS.*

A short time before the close of the last century the illustrious author of "Cosmos" wrote:* "Le nom de Galvani ne périra point; les siècles futurs profiteront de sa découverte, et, comme le dit Brandes,† ils reconnaîtront que la physiologie doit à Galvani et à Harvey ses deux bases principales." This is saying much, but, as I believe, it is not saying more than what is now fully borne out by the facts; and on this account I think it will not be waste of time to take a cursory glance at the history of the discovery of animal electricity before proceeding to deal with problems in which, as I hope to show before I have done, this agent supplies us with the master key.

The discovery of animal electricity dates as far back as 1786. In the course of this year, while amusing himself with an electrical machine, it occurred to Galvani that the hind limbs of frogs

* "Expériences sur le galvanisme, et en général sur l'irritation des fibres musculaires et nerveuses." Traduit par J. F. N. Jadelot. 8vo Paris, 1799, p. 361.

† "Versuch über die Lebenskraft." Hanover, 1795, p. 82.

might be serviceable as electroscopes in some investigations on atmospheric electricity with which he was then engaged. Some of these limbs, which were being prepared for purposes of cookery in another part of the same room, were thrown into a state of contraction whenever he drew a spark from the conductor; and this fact led him to think that discharges of atmospheric electricity might make themselves known by means of similar contractions. With the help of his nephew, Camillo Galvani, he proceeded to put this idea in practice without delay. The time was a clear and calm evening in September—an evening in which the sky was free from all signs of electrical disturbance. The place was a high terrace belonging to the house at Bologna in which Galvani lived—then the Casa Panfili-Colonna, now the Casa Monti, in the Strada S. Gervasio. Each pair of limbs was suspended by a small iron hook from the horizontal bar of the iron railings which fenced in the highest part of the terrace, the hook transfixing the portion of spine which had not been cut away. The house, the terrace, the railings, are still to be seen at No. 96 in the Strada S. Felice, the only change of moment being in the name of the street. Galvani says—"Ranas itaque consueto more paratas uncino ferreo earum spinali medulla perforata atque appensa, septembris initio (1786) die vesperascente supra parapetto horizontaliter collocavimus. Uncinus ferream laminam tangebat; en motus in rana spontanei, varii, haud infrequentes! Si digito uncinulum adversus ferream superficiem premeretur, quiescentes excitabantur, et toties ferme quoties

hujusmodi pressio adhiberetur."* How, then, were these contractions to be accounted for? Were they analogous to the contractions brought about by drawing a spark from the conductor of an electrical machine? This could scarcely be, for the sky at the time did not exhibit the necessary electrical disturbances. Could there be electricity in the limbs themselves, and were the contractions the consequences of the workings of this agent? Were the contractions arguments in favor of the existence of animal electricity? Galvani had no hesitation in answering these questions in the affirmative. Nor is this to be wondered at; for it must be remembered that frictional electricity and atmospheric electricity, neither of which had to do with the contractions, were the only kinds of electricity of whose existence he was then aware. From this time until the day of his death, Galvani went on performing experiment after experiment, sacrificing hecatombs of frogs, always firm in his belief in animal electricity, and unceasingly striving to bring others to the same mind with himself. He was, however, destined to be foiled, and that, too, by a weapon which lay hid in one of his own experiments. The experiment in question was one in which a galvanoscopic frog† was thrown into a state of momentary contraction by placing a conducting arc, of which one-half was silver and the other half copper, between

* "De Viribus Electricitatis in Motu Musculari Commentarius." 1791.

† The *galvanoscopic frog* was prepared from the hinder half of the animal, by stripping off the skin, and cutting away all the parts between the thighs and the fragment of the spine, except the principal nerves.

the lumbar nerves and the crural muscles. Galvani, as was his wont, explained these contractions by supposing that the conducting arc had served to discharge animal electricity, and that the contractions were the result of the discharge. Volta, on the other hand, was of opinion that the electricity producing these contractions originated in certain reactions between the silver and copper portions of the conducting arc; and he was not shaken in this opinion by what he did afterward, for, wishing to confirm it, he began a series of investigations which ended in the discovery of the voltaic pile and battery—a discovery which filled all minds with wonder, and for a long time afterward diverted attention altogether from the consideration of the claims of animal electricity. In the mean time, however, while Volta was demonstrating the existence of that electricity which originates in the reaction of heterogeneous bodies, and which is now known as voltaic electricity, Galvani continued his search after animal electricity, and made many important discoveries as he went along. He discovered, among other things, that a galvanoscopic frog would contract without the help of a conducting arc composed of heterogeneous metals. He discovered, not only that these contractions would happen when this arc was composed of a single metal, but also that an arc composed of muscle or nerve would answer the same purpose as the metallic arc. He also discovered that the limb of a galvanoscopic frog, of which the nerve had been divided in the loins, would contract at the moment when the end of the nerve below the line

of division was brought down and made to touch a part of the trunk of the same nerve. At last, indeed, he hit upon an experiment in which he seemed to have to do with an electricity other than that arising from the reaction of heterogeneous bodies—an electricity which must belong to the animal tissues themselves. He did much, but he did not do enough to win the battle in which he was engaged, for Volta still kept his position, denying the existence of animal electricity, and maintaining that the electricity which produced the contractions in the galvanoscopic frogs was always due to electricity arising in the reaction of heterogeneous bodies of one kind or other—silver and copper, metal and organic tissue, muscle and nerve, nerve in one state with nerve in another, as the case might be.*

In 1799, Alexander von Humboldt took up the question at issue between Galvani and Volta, and published a work† in which he showed very plainly that there was error on both sides—that Volta was wrong in ignoring altogether the influence of animal electricity in Galvani's experiments, and that Galvani was not less wrong in recognizing nothing but this influence. This he did by means of many new and curious experiments, in some of which are to be found for the first time, as it seems, a real proof that the influence causing contraction in some of Galvani's experiments was of an electrical character. In one experiment, for example, the hind limb of a frog, with the greater part of the muscles of the thigh cut away, and with a long portion of the ischiatic nerve remaining in attachment—a

* "Ann. de Chim.," t. xxiii. p. 276 and 301. † "Op. cit."

preparation very similar to that which is now known among physiologists under the name of the rheoscopic limb,* is placed upon a dry plate of glass, with a small silver coin under the free end of the nerve, and after this a communication is made between the coin and the crural muscles, first by a piece of silver, and afterward by a piece of glass, each piece being bent previously in a suitable manner. This is the experiment; the result is this,—that the muscles are thrown into a state of contraction when the piece of silver wire is used, but not so when the piece of glass is used. The result, that is to say, is one which shows very plainly that the influence causing the contractions agrees with electricity in its relations to conducting and non-conducting bodies. Humboldt, moreover, describes other experiments, to which I shall have to refer presently, which give additional probability to the same conclusion by showing that the influence causing the contraction, like electricity of high tension, is capable of acting at a distance,—of acting, so to speak, across a gap.

In 1803, Aldini, Galvani's nephew,† published an account of certain experiments to which I have also to direct attention in another part of the present lecture. These experiments furnish further evidence in favor of the existence of animal electricity by

* A *rheoscopic limb* (ῥέω, fluo; σκοπέω, exploro) is the leg of a frog, skinned, and with all the parts of the thigh cut away, except the principal nerve.

† "Account of the late Improvements in Galvanism, with a series of curious and interesting experiments performed before the Commissioners of the French National Institute, and repeated in the Anatomical Theaters of London, &c." 4to. London, 1803.

showing that living animal tissues are capable of giving rise to attractions and repulsions which seem to be no other than electrical attractions and repulsions; but, unfortunately for physiological science, the discovery of the voltaic battery had, about this time, given the victory to the opinions of Volta—a victory so complete that nothing more was heard about animal electricity for the next thirty years.

In 1827, Nobili* brought back the subject of animal electricity to the thoughts of physiologists by discovering an electric current in the frog. He made this discovery by means of the very sensitive galvanometer which he himself had invented a short time previously—an instrument which, as perfected by M. Du Bois-Reymond, is as essential to the physiologist, as the ophthalmoscope to the oculist, the stethoscope to the physician, or even the telescope to the astronomer. Immersing each end of the coil of the instrument in a vessel containing either simple water or brine, and completing the circuit between the two vessels with a galvanoscopic frog,—the fragment of the spine being immersed in one vessel, and the paws in the other,—he found that there was a current in the frog from the feet upward, which current would cause a considerable permanent deflection of the needle,—to 30° or more if brine were used, to 10°, or thereabouts, if water were substituted for brine. Nobili supposed that this current was peculiar to the frog, and he erred in so doing: he had, however, done a great thing, for, by this experiment, he had furnished another unequivocal proof of the real existence of animal electricity.

* "Bibl. Univ.," 1828, t. xxxvii. p. 10.

Twelve or thirteen years later, M. Matteucci published an essay* which, as M. de la Rive says,† "restored to animal electricity the place which it ought to occupy in electrical and physiological phenomena." This essay, moreover, had a great indirect influence upon the fortunes of animal electricity, for M. Du Bois-Reymond, as he himself tells us, was led to undertake the investigations which have made his name famous in this department of physiology by the inspiration arising from its perusal.

The joint labors of MM. Matteucci and Du Bois-Reymond have left no room for entertaining any doubt as to the reality of animal electricity. This will appear sufficiently in the sequel, when many of the experiments which furnish the demonstration will have to be referred to particularly. In the mean time, it may be said that M. Matteucci has demonstrated in the most unequivocal manner that animal electricity is capable of decomposing iodide of potassium, and of giving "signes de tension avec un condensateur délicat,"‡ as well as of producing movement in the needle of the galvanometer; and not only so, but also—a fact, the discovery of which will always give M. Matteucci a place in the very foremost rank of physiological discoverers—that muscular contraction is accompanied by an electrical discharge analogous to that of the Torpedo. And

* "Traité des Phénomènes Electro-physiologiques des Animaux." Paris, 1844.

† "A Treatise on Electricity, in theory and practice. Translated by C. V. Walker." 8vo. Longman, 1853–1858.

‡ "Cours d'Electro-physiologie." Paris, 1858.

as for M. Du Bois-Reymond* it may be said that he has demonstrated that there are electrical currents in nerve—in brain, spinal cord, and other great nervous centers, in sensory, motor, and mixed nerves, in the minutest fragment as well as in masses of considerable size,—that the electrical current of muscle, which had been already discovered by M. Matteucci, may be traced from the entire muscle to the single primitive fasciculus,—that Nobili's "frog current," instead of being peculiar to the frog, is nothing more than the outflowing of the currents from the muscles and nerves,—that the law of the current of the muscle in the frog is the same as that of the current of the muscles in man, rabbits, guinea-pigs and mice, in pigeons and sparrows, in tortoises, lizards, adders, slow-worms, toads, tadpoles and salamanders, in tench, in freshwater crabs, in earth-worms—in creatures belonging to every department of the animal kingdom,—that the law of the current in muscle agrees in every particular with the law of the current in nerve, and also with that of the feeble currents which are met with in tendon and other living tissues,—and that there are sundry changes in the current of muscle and nerve under certain circumstances, as during muscular contraction, during nervous action, under the influence of continuous and interrupted galvanic currents, and so on, which changes, as I shall hope to show in the sequel, are of fundamental importance in clearing up much that would otherwise be impenetrable

* "Untersuchungen über Thierische Electricität." Berlin, 1849, 1853.

darkness in the physiology of muscular action and sensation.

It is time, however, to leave these preliminary matters, and to proceed to the consideration of the physiological problems which wait for solution.

(2) *ON THE ELECTRICAL PHENOMENA WHICH BELONG TO LIVING MUSCLE AND MOTOR NERVE DURING THE STATE OF INACTION.*

¶ 1. *During the state of inaction the natural state of living muscle and nerve is one in which the longitudinal and transverse surfaces of the fibers are in a state of electrical antagonism, the longitudinal surfaces being electrified positively, and the transverse surfaces negatively.*

When a portion of the living muscle or nerve is included in the circuit of a suitable galvanometer,* with the longitudinal surface of the fibers in connection with one end of the coil, and with the transverse surface in similar relation to the other end, the needle of the instrument gives evidence of a current in a direction which shows that the longitudinal surface of the fibers is electrified posi-

* My galvanometer was made by Mr. Becker (Elliott Brothers, 30 Strand), after the pattern of the one used by M. Du Bois-Reymond. The coil is composed of 1 lb. 11 oz. of wire, gauge No. 32; the layers of the coil are 154; the number of coilings are 20,020, or upwards of three English miles. The needles are cylindrical, with each end sharpened out into a long point; the connecting piece is made of aluminium instead of tortoiseshell, as in M. Du Bois-Reymond's instrument—a difference by which the astatic system becomes a little lighter, 4·5 grains instead of 4·9 grains. At first I used the electrodes used by M. Du Bois-Reymond,—platinum plates immersed in a saturated solution of common salt; lately I have preferred the electrodes recommended by M. Jules Regnault, and adopted by M. Matteucci,—plates of amalgamated zinc immersed in a saturated solution of sulphate of zinc.

tively, and the transverse surface negatively. And this is the constant rule, except under certain circumstances which will have to be mentioned presently. In the case of a nerve, it is necessary to make an artificial section in order to bring out the electrical antagonism of the longitudinal and transverse surfaces. In the case of the muscles this is not necessary, for the tendinous tissue into which the ends of the fibers are inserted is found to exhibit the negative electricity of the transverse surfaces. At the same time, the tendon greatly obscures, and in some cases altogether hides, the true electrical relations of the ends of the muscular fibers inserted into it; and in all cases it is necessary to make a transverse section of the muscular fibers themselves, in order to bring out to the full the electrical relations of their transverse surfaces. In an electrical point of view, there is no difference whatever between muscular fiber and nerve fiber. In an electrical point of view, there is no difference whatever between different kinds of muscular fiber, or between different kinds of nerve fiber. These facts are abundantly established by the investigations of M. Du Bois-Reymond, and they are not now called in question by any one.

In order to explain this electrical antagonism between the longitudinal and transverse surfaces of the fibers of living muscle and nerve, M. Du Bois-Reymond supposes that these fibers are composed of what he calls *peripolar molecules*,—of molecules, that is to say, with the negative electricity gathered around the poles which point to the ends of the fiber, and with the positive electricity arranged as

an equatorial belt between the polar regions occupied by the negative electricity—an arrangement which is represented in the accompanying figure (Fig. 1) by making the parts of the molecules which

FIG. 1.

are electrified negatively dark, and the part which is electrified positively light. In this way, as M. Du Bois-Reymond supposes, the longitudinal surfaces of the fibers will present signs of positive electricity, because the equatorial belts of positive electricity around the molecules composing the fibers are turned in this direction; and the transverse surfaces of the fibers will give signs of negative electricity, because the negative poles of the component molecules will be laid bare by a transverse section of the fibers. And this may be the true view of the matter. As it seems to me, however, it is more easy to suppose that each living fiber of nerve and muscle has two sets of electrical molecules: one set in which the positive electricity is external, and the negative electricity internal; the other set in which the negative electricity is external, and the positive electricity internal—an arrangement which is shown in Fig. 2 and Fig. 3 by the same differences of dark and light shading which were used in Fig. 1 to distinguish between

positive and negative electricity, and which will be used in all future figures in which the same differences have to be marked. As it seems to me, it is

Fig. 2. Fig. 3.

more easy to suppose that the molecules in which, as in Fig. 3, the negative electricity is external, are arranged in the core of the fiber, and that the molecules in which, as in Fig. 2, the positive electricity is external, are clustered together as a coating around this core. In this way, as is seen in Figs. 4, 5 and 6, the longitudinal surface of the fiber will be electrified positively, seeing that it is composed of mole-

Fig. 4.

Fig. 5.

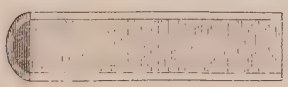


Fig. 6.

cules of which the positive electricity is external; and the transverse surface will be electrified negatively, for this surface involves the exposure of molecules of which the negative electricity is external.

Why the molecules, or why the fibers composed of these molecules, should preserve their particular electrical arrangement, is a problem of no easy solution. It may be, perhaps, that there is some physical constitution in the molecules or fibers not unlike that which exists in magnetic bodies, and which keeps separate the positive and negative elements in these bodies. But, be the explanation what it may, the fact remains, that, under ordinary circumstances, the longitudinal surfaces of the fibers of living muscle and nerve are electrified positively, and the transverse surfaces negatively: and it is upon this fact, and not upon its explanation, that I wish to insist at present.

¶ 2. *During the state of inaction the longitudinal and transverse surfaces of the fibers of living muscle and nerve present different degrees of electric tension at different points, the longitudinal surface being most positive at the point most removed from the transverse surface, and the transverse surface most negative at the point most removed from the longitudinal surface.*

This fact, which is made out by means of the galvanometer, is not unintelligible, if the electric constitution of the fibers of living muscle or nerve be that which is represented in Figs. 4, 5, and 6; for, on looking at these figures, it can easily be supposed that the positive and negative electricities of the longitudinal and transverse surfaces of the fibers will react and neutralize each other at the line of junction between these surfaces, and that the effect of this reaction and neutralization will diminish as

the distance on each side of the line of junction increases.

¶ 3. *Under certain circumstances the longitudinal and transverse surfaces of the fibers of living muscle and nerve have their electrical relations reversed, the longitudinal surface becoming negative, the transverse surface positive.*

This reversal, which is represented in Figs. 7 and 8, is found to take place a short time before the

Fig. 7.

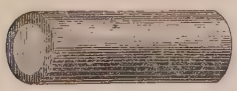
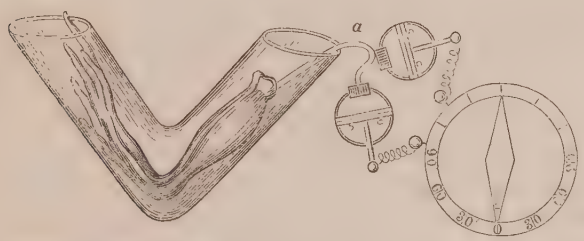


Fig. 8.

occurrence of rigor mortis in the muscles of warm-blooded animals and of certain reptiles, and in the brain and spinal cord of frogs. In nerves, also, it is found to result from various injuries—mechanical, thermal, chemical, and others. In one experiment, for example, the nerve of a rheoscopic limb is included in the circuit of a galvanometer, as is shown in Fig. 9; and then, after having waited until the

Fig. 9.



needle of the instrument has taken up the position into which it diverges under the current proceeding from the nerve into the coil, a small rod of hot iron

is brought near to the nerve at the point *a*, but not so near as to damage the nerve permanently. Before the action of the heat upon the nerve, the needle diverges in a direction which shows that the longitudinal surface of the nerve is positive, and the transverse surface negative; and it remains divergent at a given point, say at 30°. After the action of heat upon the nerve, the needle has passed to the other side of zero, and taken up a permanent position on this side, say at 10°. In other words, the action of the heat is to reverse the relative electric antagonism of the two surfaces of the nerve. It is found also that this reversal may pass off, and that the vital properties of the nerve are not materially affected so long as it lasts. Thus, it is a fact that the nerve will recover its natural electrical relations when it is placed for a short time among the moist muscles of the leg to which it belongs, or in any other place where it can recover the natural moisture which has been dissipated by the heat; and it is also a fact that the muscles of the leg may very readily be thrown into a state of contraction by acting upon the nerve, while the needle of the galvanometer shows very plainly that the natural electrical relations of the longitudinal and transverse surfaces of the nerve are reversed. The possibility of this reversal will be found eventually to be a fact of great significance: in the mean time, I merely mention the phenomenon as a fact to be remembered.

¶ 4. *During the state of inaction the fiber of living muscle and nerve presents unmistakable signs of*

current electricity if two points of dissimilar electricity or of dissimilar electric tension be included in the circuit of the galvanometer, but not so if the two points thus included are similar in electricity, or similar in electric tension.

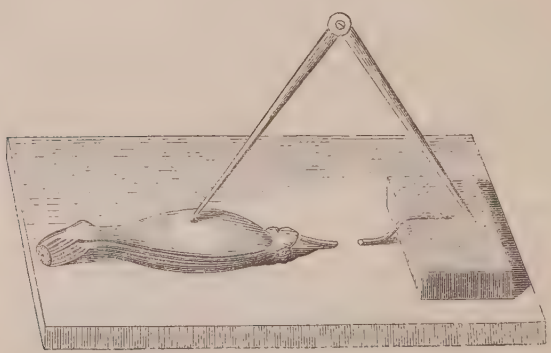
The needle of the galvanometer, that is to say, gives evidence of a current if the coil be interposed between the positive longitudinal surface and the negative transverse surface. It also gives evidence of a current if the ends of the coil be placed at points on the longitudinal or transverse surfaces which are not equidistant from the central point; for, although one surface is electrified with only one kind of electricity, the electric tension of the surface is not the same except at points which are equidistant from the central point. But the needle of the galvanometer remains motionless, and any other evidence of a current is altogether wanting, if the ends of the coil are applied to two points on the longitudinal or transverse surface which are equidistant from the central point, and in which, for this reason, the electric tension of the surface is equal. All this, as well as all that of which mention has been made hitherto, is established in the most satisfactory manner by Professor Du Bois-Reymond.

¶ 5. *During the state of inaction living animal tissues are found to be capable of acting upon the gold-leaf of an electroscope and of furnishing other signs which show that the natural electricity of these tissues is characterized by high tension.*

Electroscopic signs of animal electricity have been detected by several observers,—by Gardini and

Hemmer about the time of Galvani's great discovery, by Ahrens in 1817, by Nasse in 1834, and by myself the other day. Ahrens, using a common Bennett's gold-leaf electroscope, with a condenser, ascertained, among other things, that all parts of the body furnish signs of free positive electricity, especially when the circulation is excited in any way, and that these signs disappear under the action of great cold and in rheumatism; and these observations have been repeated and confirmed by Nasse and myself. There are also some very remarkable experiments by Humboldt, Aldini, and M. Matteucci, which show very plainly that animal electricity is characterized by considerable tension. In one of Humboldt's experiments, the leg of a very vigorour female frog is prepared and arranged as in Fig. 10; and when this is done, the space between the

Fig. 10.

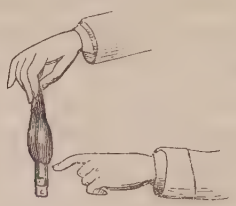


crural muscles and a small piece of metal under the outermost end of the sciatic nerve is bridged over by a pair of metal compasses. The leg, the two portions of nerve, and the small piece of metal are all made

to rest upon a perfectly dry plate of glass. The arrangement, it will be seen, is one in which the circuit is broken by a gap in the course of the nerve, for the supporting plate of glass at the gap is made perfectly dry. At the moment when the compasses are placed in the position shown in the figure, the muscles of the leg, notwithstanding the gap in the circuit, are made to contract. At first, the contraction will happen although this gap be as wide as four-fifths of a line; in the course of ten minutes or thereabouts, the contraction will not happen unless the two portions of the divided nerve are brought into actual contact. In fact, the width of the break in the circuit which is sufficient to prevent these contractions is found to become narrower and narrower in direct proportion to the loss of vitality in the nerve. Now, if the influence causing these contractions be the electricity which is inherent in the living animal tissues this electricity must be of considerable tension, for otherwise it would be incapable of acting across a gap in the circuit which would be sufficient to interrupt altogether the passage of a tolerably strong galvanic current; and that the influence in question is in reality the electricity of the animal tissues is shown in the fact—this among other proofs furnished by Humboldt—that its action is permitted by conductors and prevented by non-conductors in precisely the same way as that in which the action of electricity is permitted and prevented. The experiments of Aldini, to which I have referred, bring to light the existence of a very remarkable kind of attraction in animal bodies—an

attraction which would seem to show the presence of electricity of high tension in these bodies. "I held," says Aldini, "the muscles of a prepared frog in one of my hands, moistened by salt and water, and brought a finger of the other hand, well moistened, near to the crural nerves. *When the frog possessed a great deal of vitality, the crural nerves gradu-*

Fig. 11.



ally approached my hand, and strong contractions took place at the moment of contact." And again: "Being desirous to render this phenomenon more evident, I formed the arc by applying one of my hands to the spinal marrow of a warm-blooded animal, while I held the frog in such a manner that its crural nerves were brought very near to the abdominal muscles. By this arrangement, the attraction of the nerves of the frog became very evident. I performed this experiment for the first time at Oxford, before Sir Christopher Pegge and Dr. Bancroft, and repeated it in the anatomical theaters of St. Thomas's and Guy's Hospitals." Nor is a different conclusion to be drawn from the experiment which still remains to be noticed,—an experiment by Professor Matteucci, of Pisa, in which a "muscular pile" is formed by cutting and arranging a number of frogs' thighs

as they are cut and arranged in Fig. 12. For in addition to supplying a current to the galvanometer

FIG. 12.

of which the strength is directly related to the number of elements entering into its composition, this pile has the power of decomposing iodide of potassium, and of giving (this is the point of present interest) "*des signes de tension avec un condensateur délicat.*" This pile, in a word, is capable of furnishing the most unmistakable evidences of animal electricity.

¶ 6. *The natural electricity which is present in living muscle and nerve during the state of inaction is altogether absent in rigor mortis.*

The evidences of natural electricity take their departure *pari passu* with the evidences of that property of muscle and nerve which is usually called irritability; and the evidences of electricity and irritability are alike absent in rigor mortis. Of this fact there need be no doubt, and there is no doubt.

¶ 7. *There is reason to believe that the primary electrical condition of living muscle and nerve during the state of inaction is that of statical electricity, and that the "muscular current" and the "nerve current" which may pass from the muscle or nerve during the state of inaction are only secondary phenomena.*

M. Du Bois-Reymond is of opinion that strong currents circulate in closed circuits around each one of his peripolar molecules, that these strong currents move in the direction of the arrows in Fig. 1, and that the "muscular current" or "nerve current" which passes into the galvanometer, when living muscle or nerve is included in a particular way within the circuit of the instrument, are only *derived* portions of the strong currents moving in closed circuits, the weakness of the derived current being in no sense a measure of the strength of the primary closed current. But it is quite plain that other consequences may be deduced from this theory of peripolar molecules besides those which are deduced by M. Du Bois-Reymond. It is quite plain that these peripolar molecules must be in a state of mutual repulsion—if they are arranged as this theory requires them to be, for, as is seen in Fig. 1, this arrangement is one in which positive electricity is opposed to positive electricity, and negative electricity to negative electricity. It is quite plain, indeed, that the arrangement is one which must keep the molecules in a state of mutual repulsion; for it is a law of electricity, that similar electricities repel and dissimilar electricities attract each other. Nor is a different conclusion to be drawn from that view of the electrical constitution of the fibers of living muscle and nerve which I have suggested in paragraph 1, and which is illustrated by Figs. 2, 3, 4, 5, and 6. For what is the case here? The case is simply this: that the molecules of the core of the fiber will be kept in a state of mutual repulsion, because they are all electrified similarly with negative

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electricity; and that the molecules of the coating will in like manner repel each other, because they are all similarly electrified with positive electricity. In the sequel, I think I shall be able to show that the physiology of muscular motion and sensation will be greatly simplified by supposing that the primary electrical condition of living muscle and nerve is one of statical, and not one of current electricity—that, in fact, I have hitherto only puzzled myself in vain, and perplexed those who have listened to me, by looking upon the current as the primary condition. In the mean time, I will only say that this view will account equally for the "muscular current" and "nerve current," and for the signs of tension which have been described. The view will account for the "muscular current" and "nerve current;" for, as has been already seen, all that is necessary to obtain these currents is to bring the ends of the coil of the galvanometer into relation with two points of dissimilar electricity or dissimilar electric tension. And it will also account for the signs of tension which have been described; for tension is the grand characteristic of statical as contradistinguished from current electricity. Indeed, these very signs of tension may be appealed to as a proof that the primary electrical condition of the living muscle and nerve is statical; for it is altogether impossible to suppose that these signs can belong to a current so utterly feeble as is the "muscular current" or the "nerve current"—a current which requires a fine wire of miles in length for its detection.

¶ 8. *It is possible that the elongated state of the fibers of living muscle may be due to the presence of the*

electricity which is inherent in them during the state of inaction.

If, as has been supposed, the primary electrical condition of living muscular fiber is one of statical electricity, it follows, as a natural consequence, that the fiber will be kept in a state of elongation by the electricity. For what is the case but this? That, so long as the molecules of the core of the fibers are electrified similarly with negative electricity, they must repel each other. That, so long as the coating of the fibers are similarly electrified with positive electricity, they also must repel each other. That, so long as the molecules of the core and coating are electrified with different electricity, there must be a state of attraction between the molecules of the core and coating—an attraction which must operate chiefly across the fibers. The case, indeed, is one in which the necessary reactions of the natural electricity of the living muscular fiber would seem to involve that very change which distinguishes the elongated from the contracted state—namely, increase of length with decrease of breadth: for the electric tension of the molecules of the fibers acts without impediment longitudinally, but is resisted and neutralized laterally by the attraction which operates between the positive molecules of the coating and the negative molecules of the core.

¶ 9. *It is possible that the state of the muscular fiber in rigor mortis may be the necessary consequence of the extinction of the natural electricity of the fiber.*

This possibility is involved in the last; for if the

presence of its natural electricity causes the muscular fiber to increase in length and decrease in breadth, it follows that the absence of this electricity may bring about the opposite state of things,—namely, decrease of length and increase of breadth.

LECTURE II.

In my first lecture I began what I have to say upon the physiology of muscular motion by directing your attention to some of the electrical bearings of the problem.

I came to the conclusion that the natural electricity of living muscle and nerve is, during the state of inaction, in a statical and not in a current condition; and I ventured to say that the electrical current which passes into the galvanometer from muscle and nerve at this time, and about which so much has been said of late, is a secondary and not a primary phenomenon.

I also came to the conclusion that the presence of the natural electricity of the muscle may give rise to the state of muscular relaxation by keeping the muscular molecules in a state of electrical tension; and that the absence of this electricity may bring about rigor mortis by allowing the physical attraction of the muscular molecules to come into play.

In my present Lecture I propose to continue and finish the inquiry into the action of electricity in the process of muscular motion. In doing this, I shall have to tax your patience and attention severely, but not, I trust, needlessly or fruitlessly: not needlessly, for the questions with which I have to deal are questions to which correct answers must be

forthcoming before any real insight can be gained into the process of muscular motion: not fruitlessly, for I hope to be able to show that plain and simple answers may be found readily enough if they are sought for in the right way—a way, I would here remark, which is much shorter and easier than that in which I have hitherto attempted to find them.

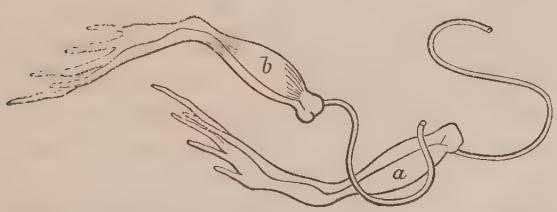
The whole subject of the action of electricity in muscular motion is, no doubt, difficult and complicated; but it is one which need not foil us, and, most assuredly, it is one which will abundantly repay the toil necessary to master it.

(3) *ON THE ELECTRICAL PHENOMENA WHICH BELONG TO MOTOR NERVE AND MUSCLE IN THE STATE OF ACTION.*

¶ 10. *The state of action in a muscle is accompanied by a discharge of electricity analogous to that of the torpedo.*

If, as in the accompanying figure, the nerve of the rheoscopic limb *b* be laid upon the muscles of the rheoscopic limb *a*, "induced contractions," as they

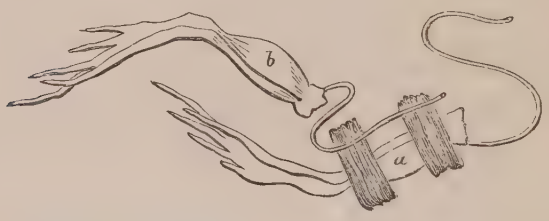
FIG. 13.



are called, will make their appearance in the limb *b* when the limb *a* is made to contract by pinching or otherwise acting upon its nerve. M. Matteucci

discovered this fact in 1842;* and M. Becquerel† explained it at the time by supposing that the contractions in the limb *a* were accompanied by an electrical discharge, and that this discharge, acting upon the nerve of the limb *b*, gave rise to "induced contraction." M. Matteucci has also a modification of this experiment in which the same fact is brought out with still greater distinctness. In this case, as is shown in Fig. 14, the nerve of the rheoscopic limb *b* is connected with the muscles of the limb *a* by means of two small pieces of lamp-cotton well

Fig. 14.



moistened in salt water. The arrangement differs in a material particular from that adopted in the last experiment, but the result is the same, for "induced contractions" are still produced in the limb *b* when the limb *a* is made to contract by acting upon its nerve. The case, indeed, is one in which the influence causing the "induced contractions" must traverse the moistened wicks of lamp-cotton, as electricity would traverse them, and as only electricity could traverse them; and thus the modification of the original experiment furnishes a strong additional reason for adopting Becquerel's

* "Traité des Phén. Electro-Phys.," &c.

† "Ann. de Chim.," t. i. 1842.

conclusion with respect to the cause of the "induced contractions."

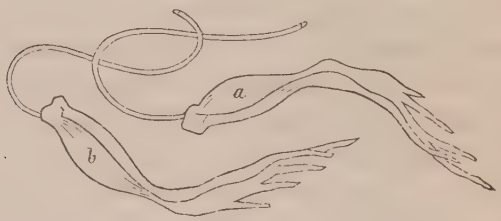
That muscular action is accompanied by a discharge of electricity in and around the muscle, is the natural inference from the last experiments, when these are taken in connection with those which have gone before. That this discharge of electricity is analogous to the discharge of the torpedo is to be inferred, as M. Matteucci has pointed out, from sundry obvious analogies between the anatomy and physiology of the muscular system and the anatomy and physiology of the electric system. For example: The nerves of the electric organs, like the nerves of the muscles, arise from the anterior track of the spinal cord, and terminate in the same loop-like plexuses. The electric organs, like the muscles, are paralyzed by the division of the nerves; and, after being thus paralyzed, both organs may be made to display their characteristic functions by "irritating" the nerve below the line of division. The electric organs and the muscles are similarly affected by strychnia, at least so far as this, that the poison gives rise to electric storms in the one case, and to convulsive paroxysms in the other. The electric organs, like the muscles, are exhausted by exercise, and require rest before they recover their power of action. And, lastly, the half exhausted nerves of the two organs respond in the same manner to the "inverse" and "direct" galvanic current (a matter of which more has to be said presently) if discharge be taken as the equivalent of contraction. In a word, the arguments from analogy are sufficiently strong to justify the con-

clusion at which M. Matteucci has arrived—that muscular action is accompanied by a discharge of electricity analogous to that of the torpedo.

¶ 11. *The state of action in a motor nerve is accompanied by a discharge of electricity which is analogous to that of the torpedo.*

If, as M. Du Bois-Reymond has shown,* the *nerve* of the rheoscopic limb *b* be laid upon the *nerve* of the rheoscopic limb *a*, the limb *b* will pass into a state of

Fig. 15.



"induced contraction" when the limb *a* is made to contract by acting upon its nerve in the usual way. As with the muscle in the two last experiments, so with the nerve in this, there is the same proof that the state of action is accompanied by a discharge of electricity analogous to that of the torpedo. Eventually, this experiment will be found to shed much light upon the nature of nervous action in general; and even now it is possible to see that it may afford a key to the explanation of what is very difficult to explain in any other way, namely, the cause of the discharge of the torpedo. For if the action of an ordinary nerve is accompanied by a discharge of

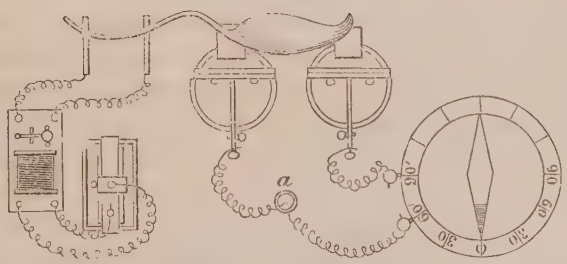
* "Untersuchungen," &c., t. ii. p. 480.

electricity, the discharge of this fish may be nothing more than the necessary consequence of the action of the nerves of organs which are arranged specially for the purpose of multiplying and intensifying the discharge. It may be, in fact, that the discharge of the nerves of the electric organs, which discharge might be insensible under ordinary circumstances, becomes multiplied and intensified by the cells of these organs, in much the same way as that in which a feeble current is multiplied and intensified by the reaction of the coils of the galvanometer.

¶ 12. *The natural electricity which is present in living muscle during the state of inaction is almost or altogether absent in the state of action.*

In the fundamental experiment by which M. Du Bois-Reymond establishes this very important fact,* the gastrocnemius of a frog is included in the circuit of a galvanometer, and the nerve belonging to the muscle is placed across the poles of an induction coil,

Fig. 16.



as is shown in Fig. 16. The experiment itself is divided into three stages. In the first stage, the

* "Untersuchungen," &c., vol. ii. pp. 50, 59.

circuit of the galvanometer is closed at *a* (where is a contrivance by which it may be closed and opened readily), and the induction coil is not in action, and the result is, that the needle moves to the right, and takes up a position—perhaps at 90°. The needle, that is to say, moves in this manner, and takes up this position, under the action of the current proceeding from the *relaxed* gastrocnemius. In the second stage of the experiment, all things being as they were at the end of the first stage, the muscle is made to contract by putting the induction coil in action, and the result is that the needle immediately swings back, and passes for a moment or two to the other side of zero—perhaps to 30°. It seems as if the current passing from the relaxed gastrocnemius into the coil is *reversed* when the muscle is made to pass from the state of inaction into that of action. In the third stage of the experiment, two things have to be done. The first thing is to shut off the current proceeding from the muscle into the galvanometer by breaking the circuit at *a*, and after this to wait until the needle has come to rest at zero. The next thing is to throw the muscle into a state of contraction by putting the induction coil in action, and then, without loss of time, to close the circuit of the galvanometer at *a*. What is done in this third stage of the experiment is to admit the current proceeding from the *contracting* gastrocnemius into the galvanometer when the needle is resting at zero. What happens in this stage is this,—that the needle moves *in the same direction* as that in which it moved under the current proceeding from the *relaxed* muscle, but *not to the same distance* from zero—to 10° perhaps,

instead of to 90°. What happens at this time, indeed, goes to show, not that the current proceeding from the relaxed muscle is *reversed* during contraction, but that this current is simply deprived of power. In other words, it is found that the muscle loses a large portion of its natural electricity when it passes from the state of inaction into that of action. I have often verified this experiment, and I have found, in addition to what M. Du Bois-Reymond has pointed out, that the rate at which the needle moves backward in the second stage of the experiment is *less rapid* than the rate at which it moves when left to fall back from the same point under the influence of simple oscillation—a plain proof, as I take it, that the action of a reversed current has nothing to do with the backward movement in question.

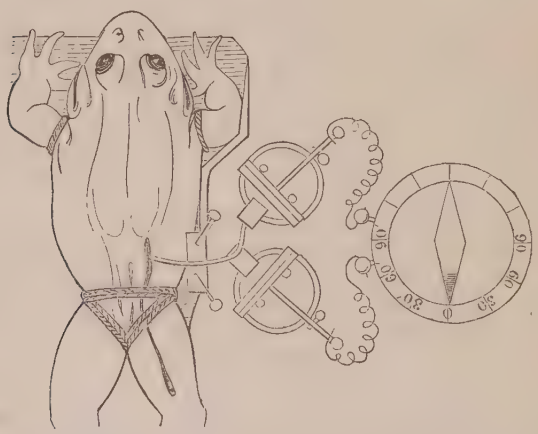
M. Du Bois-Reymond does not speak of this disappearance of electricity from the muscle during the state of action as a *discharge*. Indeed, he has his thoughts fully occupied with the idea of current electricity, and he ignores altogether the evidence of discharge which has been supplied by M. Matteucci. As it seems to me, however, M. Du Bois-Reymond supplies the very proof which M. Matteucci requires to supplement his discovery; for in the case in which M. Matteucci infers the existence of electrical *discharge*, M. Du Bois-Reymond shows that there is an actual *disappearance* of electricity.

¶ 13. *The natural electricity which is present in living motor nerve during the state of inaction is almost or altogether absent during the state of action.*

For this most important fact, as well as for the last,

physiology is indebted to M. Du Bois-Reymond.* In demonstrating it a portion of the ischiatic nerve of a frog is arranged within the circuit of a galvanometer, as is done in Fig. 17; and, after this, a few drops of a solution of strychnine are introduced under the skin of the animal. Before the poison takes effect, and while the nerve is in a state of inaction, what happens is this,—that the needle diverges under the influence of the current proceeding from the quiescent nerve, and takes up a position—say at 60°. After the poison takes effect, and when the nerve is in a state of action (for all parts of the nervous sys-

Fig. 17.



tem are then in such a state), what happens is this—that the needle returns toward zero—to 5° say, or nearer still. What happens in this latter case is, in fact, a plain proof that the natural electricity which is present in nerve during the state of inaction, is almost or altogether absent in the state of action.

* "Untersuchungen," vol. ii. p. 511.

And thus, as in the case of muscle so in the case of nerve, there is every reason to believe that the state of action is accompanied by a discharge of electricity; for, taken in connection with what has gone before, this disappearance of electricity from the nerve may be looked upon as a crucial experiment to that effect.

¶ 14. *It is possible that ordinary muscular contraction and rigor mortis may both be dependent upon the absence of the natural electricity which is present in living muscle during the state of inaction.*

Such a view, as it seems, arises naturally out of the premises: such a view, moreover, is supported by the fact that ordinary muscular contraction becomes, so to speak, confounded with rigor mortis in certain cases. Thus, in animals poisoned with strychnia, rigor mortis follows so speedily after death that its onset may readily be confounded with the last spasms of life. Thus, again, in animals killed by repeated discharges of a Leyden battery, or by repeated shocks from a Ruhmkorff's coil, there is actually no appreciable interval between the spasms preceding death and the stiffness attending death. And surely it is an argument in favor of this view, that in making ordinary muscular contraction nothing more than the momentary passage of the muscle into the unelectrified state which exists permanently in rigor mortis, it reduces ordinary muscular contraction and rigor mortis to the rule of one and the same law.

§ II. On the part which artificial electricity has to play in the process of muscular motion.

¶ 15. *Instantaneous currents of high-tension electricity, such as the discharge of a Leyden jar, or the current of a coil-machine, have the power of producing a state of action in motor nerve and muscle.*

This fact is at once familiar and indisputable and, at present, all that is necessary is to call attention to it.

¶ 16. *Continuous currents of low-tension electricity, such as the common galvanic current, have a paralyzing influence upon motor nerve and muscle.*

If the spinal cord of a rabbit be included in the circuit of a voltaic battery, and the current allowed to pass for a few moments, the part between the poles may be cut, pricked, torn, or even exposed to the shocks of a coil-machine, without giving rise either to pain or to convulsion. If a frog be poisoned with strychnia, the characteristic spasms may be averted by subjecting the spinal cord of the animal to the action of a continuous galvanic current; or if these spasms have already made their appearance, they may be at once suspended by the same means. M. Matteucci* supplies these curious facts; and, as an appendix to them, he narrates a case of tetanus in which the patient was able to open his mouth, to breathe freely, and to move his body and limbs with comparative ease, so long as a strong galvanic current was made to pass continuously along the spine from the occiput to the sacrum, or from the sacrum

* "Traité des Phén. Electro-Phys.," &c.

to the occiput. Whether the current was passed up the spine or down the spine, the result was the same, so far as its paralyzing action was concerned: and so it was also in the two experiments which have just been mentioned. There are also several beautiful experiments by Professor Eckhard, of Giessen,* which show that the action of the continuous galvanic current upon the nerve of a rheoscopic limb is to produce a state of paralysis in the part within the circuit, and that this is the case equally whether the direction of the current be up the nerve or down the nerve. In a word, there are sundry facts which show that the influence of the continuous current of low-tension electricity upon a motor nerve is altogether different to that of the instantaneous current of high-tension electricity,—that, instead of producing action, it produces paralysis.

And so also with the muscle. At any rate, M. Matteucci found that the pectoral muscle of a pigeon, from which he had dissected out all nerves of any size, could not be made to contract so long as it was acted upon by a continuous galvanic current.

At the moment of closing the circuit, and at the moment of opening it, the nerve or muscle in these experiments is (for a short time at least) thrown into a state of action; but in the interval during which the circuit is closed, and the galvanic current of low-tension is passing continuously, the part of the nerve or muscle which is actually included in the circuit is, as has been said, altogether paralyzed.

How, then, is this? How is it that the nerve of

* "Beiträge zur Anatomie und Physiologie:" Giessen, 1858. Erster Band, p. 23, &c.

a muscle, or the muscle itself, should be paralyzed by the continuous current of low-tension electricity which passes while the voltaic circuit is closed, and yet contract at the moment in which this circuit is closed or opened?

It is not difficult to account in some degree for the paralysis; for it may be supposed that the state of *polarity* which is produced by the continuous galvanic current in the paralyzed part of the nerve or muscle may derange that particular electrical state which has been seen (¶ 1) to be intimately, if not inseparably, connected with nervous and muscular irritability.

Nor is it impossible to account for the contraction which, for awhile, attends upon the closing and opening of the galvanic circuit in these experiments; for there is reason to believe that the condition of the galvanic current at these moments is widely different from that which obtains in the interval between these moments, and that the nature of the change is one which will readily account for the contraction. There is, indeed, reason to believe that the galvanic circuit is traversed at the moment of closing and opening by instantaneous currents of high-tension electricity. Thus, a stretched wire conductor along which a galvanic current is made to pass, is thrown into a state of sonorous vibration when the circuit is closed or opened, just as it is by the current of a coil-machine, or by the discharge of a Leyden jar. Thus, again, an iron wire placed in the course of a galvanic current experiences sudden changes in length and breadth when the circuit is closed and opened, just as it does when the current

of the coil-machine or the discharge of the Leyden jar is passed along it. Moreover, the spark which attends upon the closing and opening of the galvanic circuit may be supposed to lead to the same conclusion. There, is indeed, good reason for believing that the galvanic circuit is traversed by instantaneous currents of high-tension electricity at the moments of closing and opening it; and if so, then the difficulty under consideration is at an end, for it has been seen that instantaneous currents of high-tension electricity have a special power of producing a state of action in living motor nerve and muscle.

Reflecting upon these differences in the action of the instantaneous and continuous electrical current, M. Chauveau, of Lyons, who has studied these matters with great attention, has come to the conclusion that the state of action produced by electricity in nerve or muscle arises from the mechanical commotion or shock attending the current, and not from the setting-up of polar, chemical, or other changes:* and this conclusion, as it seems to me, simplifies the matter not a little, inasmuch as it brings the electrical and mechanical causes of muscular action into the same category.

¶ 17. *A motor nerve or muscle cannot be thrown into a state of action by artificial electricity unless it retain a certain amount of natural electricity.*

A muscle or nerve which has lost its natural electricity has also lost its "irritability." Of this fact there is no doubt; and, therefore, there is sufficient

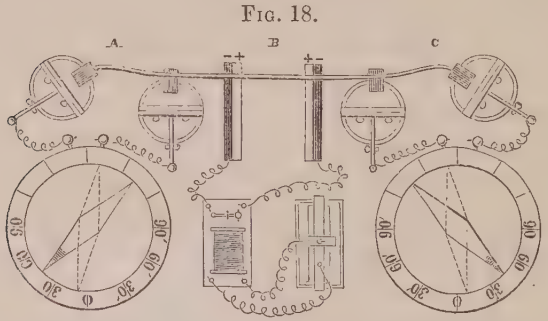
* Brown-Séquard's "Journal de la Physiologie," July and October, 1859, and January, April, and July, 1860.

reason for concluding that a certain amount of natural electricity is necessary to enable muscle or motor nerve to respond to the action of artificial electricity.

¶ 18. *When a motor nerve is thrown into a state of action by artificial electricity its natural electricity is diminished or discharged.*

M. Du Bois-Reymond* furnishes one of the proofs necessary to establish the truth of this statement by placing a long piece, *a*, *b*, *c*, of the sciatic nerve of a strong frog as it is placed in Fig. 18, that is, with its middle, *b*, across the poles of a suitable coil-machine, and its ends, *a*, *c*, each within the circuit of a galvanometer. The experiment consists in watching

Fig. 18.



the movements and position of the needles before and after the middle of the nerve, *b*, is exposed to the action of the coil-machine. Before the machine is put in action, the needle of each galvanometer moves from zero under the influence of the current proceeding from the quiescent nerve into the coil,

* "Untersuchungen," vol. ii. p. 292.

and, after oscillating for a short time, takes up a position—say at 45°: after the machine is put in action, the needle of each instrument immediately passes toward zero, and becomes stationary at 5°, or at a point still nearer to zero. This is the experiment, and this the result. In other words, the nerve is found to lose a large amount of its electricity when it is thrown into a state of action by means of artificial electricity.

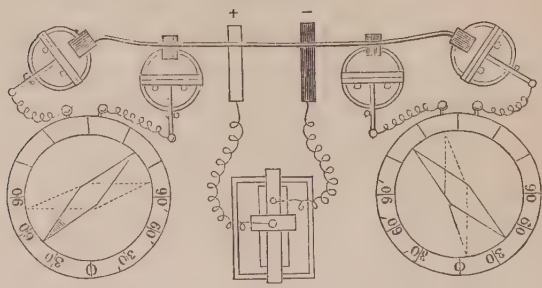
¶ 19. *When the middle of a nerve is included in a galvanic circuit, the natural electricity of the parts beyond the circuit is increased on the side of the positive pole, and diminished on the side of the negative pole.*

This statement is substantiated by an experiment of M. Du Bois-Reymond* which is the counterpart of the last, with only this difference—that instead of the middle of the nerve being placed across the poles of a coil-machine, it is here placed across the poles of a simple galvanic apparatus. Before the galvanic circuit is closed, the results are precisely the same as in the last experiment, the needle of each galvanometer diverging from zero under the action of the current proceeding from the nerve, and becoming stationary at 45° or thereabouts. After the circuit is closed, the results in the two experiments are altogether different. In the former experiment, the effect of closing the circuit of the coil-machine was to cause the needles of both galvanometers to fall toward zero—to fall into the positions indicated by

* "Untersuchungen," vol. ii. p. 292.

the dotted lines in Fig. 18: in the present experiment, the effect of closing the circuit is to move the

Fig. 19.



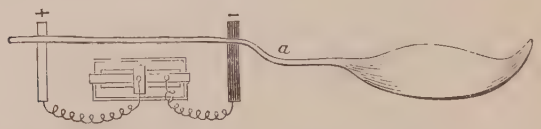
needles of the two galvanometers in opposite directions, and to bring them to a stand-still in the positions indicated by the dotted lines in Fig. 19; the needle to the right falling toward zero, and taking up a position—say at 10°; the needle to the left receding still further from zero, and becoming stationary—say at 80°. In other words, the result is one which shows most unmistakably that the natural electricity of the nerve beyond the galvanic circuit is diminished in intensity to the outside of the negative pole, and increased in intensity to the outside of the positive pole. This state of diminution and augmentation is what is spoken of by M. Du Bois-Reymond as the *electrotonic state*. The facts are indisputable; and, after what has been said, their explanation, as it seems to me, is neither impracticable nor difficult. For what is the case? The case, in the first place, is this—that the exterior of the nerve is electrified positively (¶ 1). The case, in the second place, is this—that positive electricity emanates from the positive galvanic pole, and negative

electricity from the negative. In the direction of the actual current,—that is, in the line between the poles,—the opposite electricities of the poles meet, unite, and form the galvanic current: in other directions, there is reason to believe, free negative electricity proceeds from the negative pole, and free positive electricity from the positive pole: and, therefore, what must happen under these circumstances along the nerve on the outside of each pole is sufficiently obvious. Along the nerve outside the positive pole, the positive electricity of the exterior of the nerve must be increased in intensity by the addition of positive electricity from the positive pole, and the natural electricity of this part of the nerve must be augmented accordingly; for it is to be supposed that the addition of positive electricity to the coating of the nerve will *induce* a corresponding increase in the negative electricity of the core of the nerve (¶ 1). Along the nerve outside the negative pole, on the other hand, the positive electricity of the exterior of the nerve must be weakened by the neutralizing influence of the negative electricity proceeding from the negative pole, and this weakening must involve a similar change in the natural electricity of this part of the nerve; for it is to be supposed that this weakening of the positive electricity of the coating of the nerve will, by diminishing the induction between the coating and the core, lessen in a corresponding degree the negative electricity of the core. And thus the fact which forms the subject of this paragraph would seem to meet with an intelligible explanation.

¶ 20. *When a part of the nerve of a muscle is exposed to the action of a centrifugal or "direct" galvanic current, the part of the nerve which lies between the muscular fibers and the nearest galvanic pole (the negative) is found, at the moment the circuit is closed, to lose a certain amount of electricity, and, at the same time and for a moment or two afterward, to gain a certain amount of irritability.*

That the part of the nerve which lies between the muscular fibers and the nearest galvanic pole—the part indicated in Fig. 20 by the letter *a*—should

FIG. 20.



lose electricity under these circumstances, follows from what has been said in the last paragraph (¶ 19), for it was there said that the effect of including a part of the trunk of a motor nerve in a galvanic circuit is to increase the natural electricity of the nerve outside the positive pole, *and to diminish this electricity outside the negative pole.* That the part of the nerve outside the negative pole—the part indicated in the figure by the letter *a*—does, under these circumstances, gain a certain amount of irritability at the moment when the circuit is closed and for a moment or two afterward, is not less true; for it is a fact that a drop of salt water placed upon the nerve at *a*, which drop was not sufficiently concentrated to give rise to contraction in the muscle before the circuit was closed, is strong

enough to do this after the circuit is closed. In bringing out this curious fact, the mode of proceeding adopted by its discoverer, M. Eckhard,* is as follows. In the first place, the muscle is thrown into a state of tetanus, by placing a drop of concentrated salt water upon the nerve at *a*. In the next place, this drop is diluted with water until the tetanus comes to an end for want of that degree of saltness which is necessary to keep it up. Up to this time, the galvanic circuit has been open; at this time this circuit is closed. What happened before the circuit was closed has been seen; what happens now is this—that as soon as the circuit is closed the tetanus returns. In other words, an action upon the nerve which was insufficient to keep the muscles in a state of contraction before the circuit is closed, is sufficient to do this after the circuit is closed. And hence it would seem that the gain of irritability in this case is accompanied by loss of electric tension in the nerve.

¶ 21. *When a part of the nerve of a muscle is exposed to the action of a centrifugal or "direct" galvanic current, and kept so exposed for fifteen or twenty minutes, the part of the nerve which lies between the muscular fibers and the nearest galvanic pole (the negative) is found to have lost all traces of electricity and irritability when the galvanic circuit is opened.*

This influence of the "direct" or centrifugal galvanic current in rapidly extinguishing the irrita-

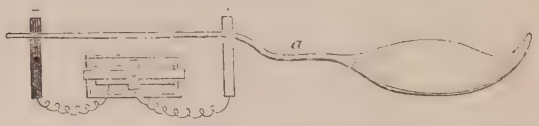
* "Beiträge," &c., Erster Band, p. 23, &c.

bility of the nerve was pointed out by Ritter* before the close of the last century, and the fact has been verified since that time by many observers and in many ways. The influence of the "direct" or centrifugal galvanic current in rapidly extinguishing the electricity of the nerve is a fact for the correctness of which I am quite ready to make myself responsible. In neither case, indeed, is there the least room for doubt.

¶ 22. *When a part of the nerve of a muscle is exposed to the action of a centripetal or "inverse" galvanic current, the part of the nerve which lies between the muscular fibers and the nearest galvanic pole (the positive) is found to have its electric tension augmented and its irritability suspended.*

That the part of the nerve which lies between the muscular fibers and the nearest galvanic pole—the part indicated in Fig. 21 by the letter *a*—should have

Fig. 21.



its electric tension augmented so long as the part of the nerve between the poles is exposed to the action of the inverse galvanic current (the current with the positive pole next to the muscular fibers), is fully proved by what was advanced in a former paragraph (¶ 19); for in this paragraph it was shown that the effect of including the middle of a nerve in a gal-

* "Beweis dass ein Selbständiger Galvanismus," &c. Weimar, 1798.

vanic circuit is to diminish the natural electricity of the nerve in the part outside the negative pole, *and to increase this electricity in the part outside the positive pole.* That the irritability of the nerve is suspended in the part in which the electric tension of the nerve is augmented—the part which is lettered *a* in the figure—is proved in a very clear manner by M. Eckhard,* for this very able physiologist has ascertained that so long as the inverse galvanic circuit is closed, it is impossible to produce contraction in the muscle by pinching, pricking, or otherwise acting upon this part of the nerve—that, in fact, the result is altogether different from that which is obtained by acting upon the same part of the nerve when (as in Fig. 20) the negative galvanic pole is nearest to the muscles. In a word, the facts are such as to render it necessary to believe that the state of augmented electric tension in a nerve is associated with a state of suspended irritability in the nerve.

¶ 23. *When a part of the nerve of a muscle is exposed to the action of a centripetal or "inverse" galvanic current, and kept so exposed for two or three hours, the part of the nerve which lies between the muscular fibers and the nearest galvanic pole (the positive) is found, when the circuit is opened, to retain its electricity and to resume (even with some additions) its irritability.*

This influence of the "inverse" galvanic current in preserving the "irritability" of a motor nerve was made known by Ritter at the same time that he pointed out the contrary action of the "direct"

* "Beiträge," &c., Erster Band, p. 23, &c.

current (¶ 21), and the fact has been abundantly verified since his time: the influence of the "inverse" or centripetal galvanic current in preserving the electricity of the nerve is a fact to the truth of which I am fully prepared to bear testimony. This latter influence is, indeed, the necessary inference from the premises; for it has been seen (¶ 6) that a nerve does not retain its irritability without retaining its electricity also.

¶ 24. *When a part of the nerve of a muscle is alternately exposed to the action of a "direct" and "inverse" galvanic current, the part of the nerve which lies between the muscular fibers and the nearest galvanic pole is found to lose and recover its electricity alternately, and therefore it may be assumed that these losses and recoveries of electricity may in some measure account for the losses and recoveries of irritability which take place under these circumstances, and which are known as "voltaic alternatives."*

It is a fact for the correctness of which I can fully vouch, that the alternate losses and recoveries of irritability which result from the alternate action of the "direct" and "inverse" galvanic current upon the nerve of a muscle, are accompanied by similar losses and recoveries of the natural electricity belonging to the nerve; and therefore it may fairly be assumed that these alternations in the condition of irritability may find their explanation in the alternations of the electrical condition.

¶ 25. *When the nerve of a muscle is exposed for some time to the action of a centrifugal or "direct" gal-*

vanic current (the current in which the negative pole is nearest the muscle), the muscle contracts strongly when the galvanic circuit is closed, and not at all when the circuit is opened.

When a motor nerve is acted upon by a galvanic current, the part of the nerve which is included between the two poles is very soon paralyzed by the current (¶ 16). When a motor nerve is acted upon by a galvanic current, the effect of this action upon the part of the nerve which lies between the muscle and the nearest galvanic pole is one thing if this pole be the positive, and another thing if this pole be the negative (¶¶ 20, 21, 22, 23). It would seem, indeed, that the action of either pole taken singly upon the irritability of a motor nerve is not only different to the action of the other pole taken singly, but that these two actions are mutually counteractive and antagonistic. When, therefore, the nerve of a muscle is *exposed for some time* to the action of a centrifugal or "direct" galvanic current (a current in which the negative pole is nearest the muscle), and the muscle contracts strongly when the circuit is closed, and not at all when the circuit is opened, I may assume two things. I may assume that the part of the nerve which lies between the two poles has become paralyzed by the galvanic current: I may also assume that the effect of this paralysis will be, so to speak, to keep asunder the actions of the two poles, and to leave the part of the nerve which lies between the muscle and the nearest galvanic pole to be acted upon by the *negative pole singly;* for in the case under consideration it is the negative pole

which is the nearest to the muscle. I may make this latter assumption, for nerve deprived of its irritability can neither receive impressions, nor transmit them. All this I may do: and this, as it seems to me, is all that I need do in order to be able to explain why, under these circumstances, the muscle should contract strongly at the closing of the circuit, and not at all at the opening. For what are the simple facts? The simple facts are these: that in the case where the nerve of a muscle is acted upon by a galvanic current in which the negative pole is nearest the muscle, the irritability of the part of the nerve which lies between the muscle and the pole is augmented at the moment when the galvanic circuit is closed, and suspended at the moment when this circuit is opened (¶¶ 20, 21). The facts, indeed, are precisely those which are necessary to explain what has to be explained, namely, the strong contraction at the closing of the circuit, and the absence of contraction at the opening: for the instantaneous currents of high tension which attend upon the moments of opening and closing the circuit, and which produce the contractions which occur at these moments when contractions do occur (¶ 16), must act strongly at the closing of the circuit, because then the irritability of the part of the nerve acted upon is augmented, and must fail to act at the opening of the circuit, because then the irritability of the part of the nerve acted upon is suspended.

¶ 26. *When the nerve of a muscle is exposed for some time to the action of a centripetal or "inverse" galvanic current (the current in which the positive pole*

is nearest the muscle), *the muscle contracts strongly when the circuit is opened, and not at all when the circuit is closed.*

In order to explain these phenomena, it is only necessary to remember the mode of proceeding adopted in the last paragraph, and to realize the effect of the positive pole, taken singly, upon the irritability of the part of the nerve which lies between this pole and the muscular fibers. This is the question, and this also is a question which is no sooner asked than answered, for, under these circumstances, it has been ascertained (¶¶ 22, 23) that the irritability of this part of the nerve is suspended when the galvanic circuit is closed, and augmented when this circuit is opened. The answer is, indeed, one which explains why, under these circumstances, the instantaneous currents of high tension which attend upon the moments of closing and opening.the galvanic circuit, and which produce the contractions which occur at these moments when contractions do occur (¶ 16), should fail to produce contraction when the circuit is closed, and should produce strong contraction when the circuit is opened; for when the circuit is closed, the irritability of the part of the nerve acted upon is suspended, and when the circuit is opened, this irritability is for the moment exaggerated.

¶ 27. *When the nerve of a muscle is first exposed to the action of a galvanic current, the muscle contracts moderately and equally at the closing and at the opening of the circuit, and it is immaterial, so far as these results are concerned, whether the gal-*

vanic current along the nerve be "inverse" or "direct."

When the irritable nerve of a muscle is *first* exposed to the action of a galvanic current, the part of the nerve which is included between the poles is *not* paralyzed by the action of the continuous current, as it is after a time, and as it was assumed to be in the cases considered in the last two paragraphs. When the irritable nerve of a muscle is *first* acted upon by a galvanic current, the condition of the nerve, as to irritability, is indeed such as to allow the action of each pole to be transmitted to the muscle. If, then, this be the condition of the nerve as to irritability, it follows that contraction may occur at the closing and at the opening of the circuit indifferently; for as the action of the positive pole singly is to permit contraction only when the circuit is opened, and as the action of the negative pole singly is to permit contraction only when the circuit is closed, it is evident that the combined actions of the two poles *may* issue in contraction at the closing or opening of the circuit indifferently. And it follows also that these contractions will be equal in amount and moderate in degree, and that it will be of no moment, so far as they are concerned, whether the positive pole or the negative pole be the nearest to the muscle; for so long as the condition of the nerve as to irritability is such as to allow the action of each pole to be transmitted to the muscle, it is evident, not only that the action of each pole upon the nerve will be equalized and moderated by the antagonistic action of the other pole, but also that it is practically immaterial whether the action of either pole upon

the nerve has to start from a point which is a little nearer to or a little further from the muscle.

¶ 28. *There is reason to believe that the differences in the order of contraction at the opening and closing of the galvanic circuit, of which mention has just been made, and which have been ascribed to differences in the direction of the current along the nerve—that is, to the current being "direct" or centrifugal in the one case, and "inverse" or centripetal in the other case,—are in reality to be ascribed to differences in the position of the poles, that is, to the negative pole being next the muscle in the former case, and to the positive pole being in this position in the latter case.*

All the last stages of the argument lead to this conclusion, and to this conclusion only. At any rate, I fail altogether to perceive any alternative.

¶ 29. *When a muscle is itself exposed to the action of artificial electricity, it contracts, or it does not contract, in obedience to the same law as that which is in operation when its motor nerve is acted upon in this manner.*

This matter has not been so fully investigated as it might be; but the facts, so far as they are known, bear out this statement in all particulars.

¶ 30. *The results which are obtained when a motor nerve or muscle is exposed to the action of artificial electricity appear to be the natural consequences of the reaction which must take place between the artificial electricity on the one hand and the natural electricity on the other.*

I draw this inference as that which may fairly be drawn from the evidence advanced hitherto; and, having done so, I gladly take leave of the intricate and perplexing inquiry which has formed the subject of the present and preceding Lecture.

LECTURE III.

In my last Lecture I continued the inquiry which was commenced in my first Lecture—the inquiry into the part which animal electricity has to play in the process of muscular motion,—and after considering the electrical phenomena which belong to motor nerve and muscle in the state of action, I went on to examine the *modus operandi* of artificial electricity in muscular motion.

In the first part of this subject, I showed, among other things,—

That the state of action in motor nerve or muscle is accompanied by a discharge of electricity analogous to that of the torpedo;—

That the natural electricity which is present in motor nerve and muscle during the state of inaction is almost or altogether absent during the state of action;—and

That ordinary muscular contraction and rigor mortis may both be dependent upon the absence of the natural electricity which is present in living muscle during the state of inaction.

It is impossible for me to summarize in a few words the many results arrived at in the second part of my inquiry; and I will therefore content myself with saying that all these results agree with those arrived at previously, and that they all resolve them-

selves into the natural consequences of the reactions which must take place between artificial electricity on the one hand and natural electricity on the other.

In my present Lecture I have to consider how the process of muscular motion is affected by the action of the blood, of "nervous influence," so called, and of certain other non-electrical agents which are of comparative insignificance as compared with these two.

§ III. ON THE PART WHICH CERTAIN NON-ELECTRICAL AGENTS HAVE TO PLAY IN THE PROCESS OF MUSCULAR MOTION.

(2) *ON THE PART WHICH THE BLOOD HAS TO PLAY IN THE PROCESS OF MUSCULAR MOTION.*

¶ 31. *The state of rigor mortis is associated with stagnation and coagulation of the blood.*

This fact requires no demonstration, and all that is needed now is to call attention to it.

¶ 32. *Muscles which have passed into the state of rigor mortis will return into the state of vital relaxation if their muscles be supplied with a sufficient amount of blood.*

This fact, which has not yet lost its novelty, has been abundantly demonstrated by Dr. Brown-Séquard,* and by the late Professor Stannius,† of Rostock.

* Comptes Rendus, Juin 9 et 28, 1851.

† "Untersuchungen über Leistungfähigkeit des Muskeln und Todtenstarre, Vierordts-Archiv für Phys. Heilkunde." Stuttgart, 1 Heft, 1852.

On the 12th of July, 1851, Dr. Brown-Séquard began an experiment which consisted in the injection and reinjection of a pound of defibrinated dog's blood into the principal artery of the arm of a criminal who had been guillotined at 8 o'clock on the morning of that day. The injections were commenced at 11 P.M., the arm then being in a perfect state of rigor mortis. A moment or two afterward, some reddish spots, not unlike those of measles, made their appearance, more particularly about the wrist. Then these spots became larger and larger, until the whole surface acquired a reddish violet hue. A little later, and the skin generally had acquired its natural living color, elasticity, and softness, and the superficial veins stood out distinct and full as during life. Then the muscles relaxed, and recovered their irritability, first in the fingers, lastly in the shoulder. At 11.45 P.M. this irritability was found to be more decided than it was at 5 P.M., at which time the corpse was first examined; and from 11.45 P.M. until 4 A.M., when the distinguished experimenter was obliged to succumb to fatigue, there was no alteration in this respect. When the experiment was commenced the temperature of the blood was 75° Fahrenheit, and that of the room 66°.

Another experiment was upon a full-grown rabbit which had been killed by hæmorrhage. In this case, after waiting until rigor mortis had fully set in, Dr. Brown-Séquard injected the defibrinated blood of the same animal into the principal vessel of one of the hind limbs. Fifteen minutes afterward, the muscles of this limb had lost their stiffness, and recovered their irritability. From this time, throughout the

night, and until 3 P.M. on the day following, the injections were repeated at intervals of from twenty to thirty minutes, and all this while the muscles which had relaxed were highly irritable. The experiment was discontinued from 3 P.M. to 4.50 P.M., and then resumed. On resuming it, the limb upon which the injections had been practiced was found (with the exception of a few bundles of fibers here and there) to have returned to its former stage of rigor mortis: after persevering with it for a short time, the result was the same as at first, the stiffened muscles again becoming relaxed and irritable as the blood gained free access to them. The result was also the same as at first in this—that the muscles remained relaxed and irritable so long as the injections were kept up. On the morning following, the parts of the body which had been left to themselves were beginning to pass out of the state of cadaveric rigidity, but not so the part which had been experimented upon. On the third morning, rigor mortis was still undiminished in this latter part; and, in marked contrast to this state of things, the rest of the animal was soft, and in an advanced state of putrefaction.

About the time that Dr. Brown-Séquard was engaged in these and other experiments of the kind, Professor Stannius, without any knowledge of what was being done in Paris, was carrying out an analogous series of inquiries at Rostock.

At 7.30 A.M. on the 21st of July, 1851, Professor Stannius put ligatures around the abdominal aorta and crural arteries of a puppy, and tied them. A few minutes after 10 A.M. the muscles had begun to stiffen in all the parts from which the blood was ex-

cluded. At 10.45 A.M. both hind limbs were stretched out, and perfectly stiff and cool. At 11.40 A.M. the ligatures were loosened, and the blood was seen and felt to penetrate into the empty vessels. At 11.45 A.M. the natural warmth had returned in some degree to both hinder limbs, and the right limb was a little more flexible than the left. At noon both limbs had undoubtedly recovered their flexibility, and it once appeared as if the left had moved spontaneously; but no sign of pain was caused by pinching the toes. At 12.30 P.M. the muscles which had been rigid, contracted everywhere on the application of galvanism; and at one time this application seemed to cause pain, for the animal, which was before quiet, gave a sudden plunge forward when it was made. Death happened unexpectedly at 12.45 P.M.

Early in the morning of the day following, a similar experiment was performed upon another puppy. At noon the paralyzed hinder limbs were perfectly supple, but the muscles below the knee had ceased to respond to the action of galvanism. At 2.15 P.M. both these limbs were stretched out and rigid, and all signs of irritability were at an end. At 2.35 P.M. the ligatures were untied. At 3.35 P.M. galvanism gave rise to strong contractions in the muscles of both thighs, and to weaker contractions in the muscles of the left leg below the knee; and very few traces of rigidity were to be discovered anywhere. At 5.35 P.M. the muscles, now perfectly soft and pliable everywhere, responded readily to the prick of a scalpel, as well as to the shock of a coil-machine. On the morning following, the animal was found dead.

In another experiment in which the abdominal aorta and the crural arteries of a fine puppy were tied, and left tied to the end, Professor Stannius shows very clearly that the rigidity of which mention is made in the two last experiments is identical with rigor mortis. In this case, four hours after the operation, the muscles *below* the ligatures were perfectly rigid and devoid of irritability. In the evening of the day following there was no alteration of any moment. Twelve hours later the animal was found dead, with the parts *above* the ligatures in a state of rigor mortis, and with the parts *below* the ligatures—which parts had been rigid before death—flaccid, moist, and exhaling a putrescent odor. In other words, the parts *below* the ligatures were in the state which comes on after rigor mortis; and hence it follows that the stiffness which had existed in these parts before the death of the anterior half of the animal must have been identical with rigor mortis.

Here, then, are certain experiments which would seem to show most unequivocally that muscles which have passed into the state of rigor mortis will return into the state of vital relaxation if these muscles be supplied with a sufficient amount of blood.

¶ 33. *The state of rigor mortis appears to depend in part upon the simple absence of that action of the blood which seems to have to do with the production of vital relaxation in muscle.*

This inference, as it seems to me, is the only inference which can be drawn from the facts advanced in the last two paragraphs; and, so far as I know, there are no facts remaining in the background

which are at all calculated to invalidate it. And this inference, which I had drawn from the experiments of Dr. Brown-Séquard before my attention was directed to the experiments of Professor Stannius, is the same as that which the last named physiologist has drawn from his own investigations.

¶ 34. *Increased disposition to ordinary muscular contraction appears to be associated with diminished supply of blood to the muscular system.*

Comparative anatomy supplies several facts which go to show that the organism which is least liberally supplied with blood is that in which the muscles and nerves are most irritable. For example:—It is a fact that the circulation is less active in fishes and reptiles than in birds and mammals; and it is also a fact that the muscular system is more irritable in the former classes than in the latter:—it is a fact that the circulation is less active in the involuntary muscular system of an animal than in the voluntary muscular system; and it is also a fact that irritability is a more marked phenomenon in the former system than in the latter:—it is a fact that the circulation of a hibernating animal is well-nigh at a standstill during hibernation; and it is also a fact that the muscles and nerves of such an animal are more irritable during this state than they were during the period of summer life.

There is also an interesting experiment by Professor Claude Bernard* which bears out the same conclusion by showing that the muscles and nerves

* "Leçons sur la Physiologie et la Pathologie du Système Nerveux." 8vo. Paris, 1858, vol. ii. p. 12.

of a warm-blooded animal acquire a reptilian degree of irritability when the circulation is depressed to the reptilian standard of activity. This experiment is one in which the spinal cord of a rabbit was divided between the fifth and sixth cervical vertebræ. Immediately after the operation, the animal lay helplessly on its side, panting, breathing almost exclusively by the diaphragm, passing fæces continually, and deprived, as a matter of course, of all power of feeling and voluntary movement in the limbs and trunk. A little later, it had recovered so far as to eat with avidity a carrot which was offered to it. Seven hours after the operation, the breathings of the animal were very slow and shallow. About this time, also, the paralyzed parts were cold and comparatively bloodless, and their motor nerves and muscles were in a state of greatly augmented irritability — a condition altogether different from that of the non-paralyzed parts, for, owing to the knife having trenched upon the cilio-spinal region of the cord, these latter parts, and especially the ears, were hot, bloodshot, and with the irritability of their motor nerves and muscles diminished rather than increased. Thirty minutes after death—the intermediate stages of the experiment are of no moment—the motor nerves and muscles of the parts which had *not* been paralyzed before death had ceased to manifest any signs of irritability, and here and there the muscular fibers were beginning to pass into the state of rigor mortis ; and, in marked contrast to this condition, the motor nerves and muscles of the parts which had been paralyzed before death were sufficiently irritable to make it possible to perform on

one of the hind limbs, prepared in a suitable manner, all the experiments which may be performed on the rheoscopic limb of the frog. In a word, the irritability of the muscles and nerves had changed from a mammalian to a reptilian standard in the parts in which the circulation had changed from a mammalian to a reptilian standard.

¶ 35. *General convulsion is brought about by sudden hæmorrhage.*

This fact needs no demonstration. If it do, a visit to the shambles is all that is necessary to supply all that can be required.

¶ 36. *General convulsion is brought about by suddenly arresting the arterialization of the blood.*

This fact, like the last, is well established. To die by strangling, or by any other form of sudden suffocation, is, indeed, to die in convulsion; and death from this cause implies, of necessity, sudden arrest in the arterialization of the blood.

¶ 37. *There is reason to believe that one way in which strychnia or brucia brings about spasmodic muscular contraction is by producing a change in the blood which is equivalent to loss of arterial blood.*

It has been shown by Dr. Harley* that air which has remained for some time in contact with blood to which strychnia or brucia has been added, contains more oxygen and less carbonic acid than air which has been left in contact with simple blood for the same length of time. It has been shown, that

* "Lancet," 7th and 14th June, and 12th July, 1856.

is to say, that blood poisoned in this manner *respires* less freely than pure blood. In one of the experiments by which this demonstration is effected, two tubes, similar in size and graduated upon the same scale, are filled half full of calf's blood. Then, after adding a minute portion of strychnia to the blood in one of the tubes, and after freely agitating the blood in each tube with supplies of fresh air, both tubes are corked up and set aside with the corks downward. During the next twenty-four hours, the blood and air thus corked up are occasionally well shaken together. At the end of this time, the air which has been all this while in contact with the blood is analyzed by Bunsen's method; and it is found, as is shown in the accompanying table, that the air which has been in contact with the poisoned blood contains more oxygén and less carbonic acid than the air which has been in contact with simple blood.

	Composition of common air.	Composition of air after having been in contact with *simple blood* for twenty-four hours.	Composition of air after having been in *blood containing strychnia* for twenty-four hours.
Oxygen . . .	20·96	11·33	17·82
Carbonic Air .	·002	5·96	2·73
Nitrogen . . .	79·038	82·71	79·45
	100·000	100·000	100·00

It is found, that is to say, that the former air is altered in a manner which shows that the strychnia

has worked a change upon the blood which may, in one sense, be looked upon as equivalent to loss of blood; for blood which cannot become arterial is as good as lost to all purposes of life. Nay, this change may be looked upon as equivalent to copious loss of blood; for in the experiment of which the results are given in the preceding table a very minute quantity of the poison has the effect of depriving the blood of full two-thirds of its natural power of becoming arterial. When brucia was used in place of strychnia, the only difference was one of degree, the power of preventing the arterialization of the blood being somewhat less energetic in the case of the brucia.

¶ 38. *There is reason to believe that the influence of venous blood in the process of muscular motion is equivalent to the absence of the influence of arterial blood, and to no more than this.*

Some time ago,* Dr. Brown-Séquard arrived at the conclusion that venous blood was capable of producing muscular contraction by acting as a stimulus to a vital property of irritability in nerve and muscle. He saw animals thrown into a state of general convulsion when the whole mass of their blood was rendered venous by suffocation; he saw certain muscles passing into a state of contraction when black blood was injected into their arteries; he saw other phenomena of lesser importance which seemed to have the same significance: and these facts, and especially the one first mentioned, led him to this conclusion. As it seems to me, how-

* "Philadelphia Med. Examiner," Nov. 1851.

ever, this convulsion and these contractions may be more easily explained by referring them to absence of arterial blood, than by referring them to presence of venous blood; for similar convulsion and similar contractions, fact for fact, may be produced when absence of arterial blood is the sole assignable cause. Thus, for example, an animal is not less certainly thrown into a state of general convulsion by hæmorrhage than by suffocation. And surely it is more philosophical as well as more easy to adopt this latter explanation; for in supposing that venous blood is, as it may well be, the equivalent of absence of arterial blood, a cause is obtained which is of avail, not in one case only, but in both cases equally —in the case in which convulsion and contractions are associated with presence of venous blood, and in the case in which convulsion and contractions are associated with absence of arterial blood.

¶ 39. *As in rigor mortis, therefore, so in ordinary muscular contraction, the contracted condition of the muscle would seem to be associated with the absence of that action of the blood which seems to have to do with the production of the relaxed condition in living muscle.*

All the previous evidence would seem to lead to this conclusion, and to none other. Why it should be so—why the absence of the action of the blood should bring about muscular contraction in one form or other, is another question—a question, too, which must remain in abeyance until some definite information is gained which can only be gained in the next section of our argument.

(2) *ON THE PART WHICH "NERVOUS INFLUENCE" HAS TO PLAY IN THE PROCESS OF MUSCULAR MOTION.*

¶ 40. *There is reason to believe that rigor mortis is associated with entire absence of "nervous influence."*

The nerves have lost their characteristic irritability before the occurrence of rigor mortis; the nerves have lost their proper electricity before the occurrence of rigor mortis; and, therefore, there is reason to believe that the nerves have ceased to exercise their special influence upon the muscles before the occurrence of rigor mortis. And certainly this is the conclusion which must be drawn from the condition of the common vascular system when the muscles pass into the state of rigor mortis; for with the blood stagnant and dead, how can there be any development of "nervous influence" in the nervous centers relating to the muscles?

¶ 41. *There is reason to believe that ordinary muscular contraction is associated with deprivation of "nervous influence," and not with a contrary state of things.*

There is no lack of evidence to show that muscular contraction may occur in its most exaggerated form under circumstances in which the amount of nervous influence supplied by the nervous centers to the muscles must of necessity be at or near zero.

The convulsion of hæmorrhage is a case in point —a case, too, of no doubtful significance. For if it be true, as it undoubtedly is, that the functional activity of an organ is directly proportionate to the activity of the circulation of arterial blood in that

organ, and if the function of certain nerve-centers be to supply nervous influence to the muscles, then it follows that this supply of nervous influence must be interrupted in the most effectual manner when the whole muscular system is thrown into a state of convulsion by loss of blood.

And, certainly, the inference which may be drawn from the occurrence of convulsion during hæmorrhage is confirmed in the fullest manner by certain experiments of Astley Cooper, and Drs. Kussmaul and Tenner.

"I tied," says Sir Astley Cooper,* "the carotid arteries of a rabbit. Respiration was somewhat quickened, and the heart's action increased; but no other effect was produced. In five minutes, the vertebral arteries were compressed by the thumb, the trachea being effectually excluded. Respiration stopped almost directly, convulsive struggles succeeded; the animal lost its consciousness, and appeared dead. The pressure was removed, and it recovered with a convulsive inspiration. It then lay upon its side, making violent convulsive efforts, breathing laboriously, and with its heart beating rapidly. In two hours it had recovered, but the breathing was still laborious. The vertebrals were compressed a second time; respiration stopped; then succeeded convulsive struggles, loss of motion, and apparent death. When let loose, its natural functions returned with a loud inspiration, and with breathing excessively labored. In four hours, it moved about, and ate some greens. In five hours, the vertebral arteries were compressed for the third

* "Guy's Hospital Reports," No. III. 1836.

time, and with the same effect. In seven hours, it was cleaning its face with its paw. In nine hours, the vertebral arteries were compressed for the fourth time, and the result was the same, viz., suspended respiration, convulsion, and loss of consciousness. On removal of the pressure, violent and laborious respiration ensued, and afterward the breathing became very quick. After forty-eight hours, for the fifth time, the compression was applied with the same effect."

The tale which is told by this well-known experiment appears to be, that convulsion may coexist with a state of things which involves interruption in the functional activity of the great cranio-cervical nervous centers—for such interruption must necessarily be brought about by arresting the flow of blood through the cervical arteries. And this tale is also that which is told in still plainer terms by Drs. Kussmaul and Tenner in the following experiment:*

In this experiment, the common innominate and the left subclavian arteries of a rabbit—that is to say, the only two great vessels proceeding from the arch of the aorta, for in this animal the right subclavian and both carotids usually commence in a common innominate artery, while the left subclavian springs independently from the aorta—are included in ligatures of which the knots are so arranged as to admit of being unloosed easily. In the first place, the blood is suddenly shut off from the great nerve-centers of the head and neck by tying the ligatures;

* "Untersuchungen z. Naturlehre der Menschen u d. Thiere," von I. Moleschott, vol. ii. Frankfort, 1859.

in the second place, the stream of blood is allowed to return to these nerve-centers by untying the ligatures at the end of a minute and a half or two minutes. Upon tying the ligatures, the animal immediately loses consciousness, and falls into a state of general and violent convulsion; upon untying the ligatures, the convulsion, which is then raging at its height, immediately comes to an end, and soon afterward consciousness and the voluntary power over the muscles are recovered. Upon untying the ligatures, the sudden passage from convulsion to muscular relaxation gives the impression of the animal having been struck down, at that particular moment, by a stroke of paralysis. The result, indeed, is one which appears to be only intelligible on the supposition that the convulsion is dependent upon the interruption in the supply of nervous influence which the muscles receive from certain great nerve-centers so long as these centers are kept in a state of functional activity by the continuance of the circulation.

In this experiment, indeed, and also in that by Astley Cooper related previously, the lesson to be learnt is one and the same; and this lesson is that which agrees with the seemingly paradoxical proposition which forms the heading to the present paragraph.

¶ 42. *There is reason to believe that muscles do not pass into a state of contraction when they may be supposed to receive a larger supply of "nervous influence" than usual.*

An experiment in which Drs. Kussmaul and

Tenner* tied the two subclavian arteries of a rabbit at their origin, and at the same time placed another ligature around the aorta a little below the origin of the left subclavian artery, may be cited in support of this statement. In this case the vessels are tied in a way which brings about an opposite result to that which was brought about by the plan adopted by Drs. Kussmaul and Tenner, in the experiment just mentioned (¶ 41). In the present case, the blood is cut off from the trunk and limbs, and the circulation confined to the head and neck: in the former case, the blood was cut off from the head and neck, and the circulation confined to the trunk and limbs. In the present case, indeed, it may be supposed that there will be increased development of nervous influence in the nervous centers of the head and neck, for all the blood in the body is directed toward these centers. The experiment under consideration consists of two parts. In the first part, the ligatures are tied; in the second part, the ligatures being still tied, pressure is made with the fingers upon the open vessels so as to cut off the supply of blood to the great nervous centers of the head and neck. In the first part of the experiment, what happens is paralysis in the parts from which the blood is excluded,—paralysis almost everywhere, convulsion nowhere. What happens in the second part of the experiment is general convulsion of great violence. In the second part, indeed, the result agrees with and confirms that which was arrived at in the experiment recorded in the last paragraph; for in this experiment violent general convulsion was

* Op. cit. vol. ii. 1859.

brought about by the tightening of the ligatures upon the vessels which supply blood to the great nerve-centers of the head and neck. For present purposes, however, the special interest of the experiment under consideration is in its first part, and not in its second; and this is because this first part supplies a proof which is not to be controverted—that convulsion is not produced when the condition of the circulation would seem to necessitate increased development of nervous influence in the great nervous centers of the head and neck. This it does, as it would seem, without leaving any room for doubt; and, in so doing, it furnishes a fatal objection to the notion that increased development of nervous influence, arising from "determination of blood to the head," is the cause of convulsion.

¶ 43. *There is reason to believe that the power of muscular contraction is inversely related to the amount of nervous influence supplied to the muscles from the great nervous centers.*

Two experiments, both supplied by the untiring research of Dr. Brown-Séquard,* furnish the clearest demonstration of the truth of this statement.

In the first of these experiments, one of the scales belonging to the small balances used by druggists is fixed immediately above the paw of one of the hind limbs of a frog, and the animal is then suspended by its fore paws so as to raise the scale to a convenient height above the table. Then the limb is put on the stretch by placing in the scale a weight which is just a trifle heavier than that which can be raised

* "Comptes Rendus," Mai 16, 1847.

by the contractions which are produced in the limb by pinching its toes. After this the spinal cord is divided immediately below the insertion of the nerves belonging to the fore limbs, and the power of the contractions which are produced in the weighted limb by pinching its toes is tested at different times after the operation, by taking from, or adding to, the weight in the scale. This is what is done. What happened in two experiments, which may be distinguished as A and B, is as follows:

		A.	B.
Grammes raised *before* the division of the spinal cord		60	60
Grammes raised *after* the division of the spinal cord.	Immediately afterward	20	10
	In five minutes	45	30
	In fifteen minutes	60	40
	In twenty-five minutes	80	60
	In one hour	130	100
	In two hours	140	120
	In four hours	140	130
	In twenty-four hours	150	140
	In forty-eight hours	150	140

Dr. Brown-Séquard also tells us that in many experiments of the same kind this lifting power in the paralyzed limb remained stationary for six, ten, or twenty days, and then began to decline at a rate which brought it to its original value before the operation in about a month, and to a third or fourth of this value in six, seven, or eight months; and he at the same time expresses his belief that some part of this failure in power might have been prevented if sufficient care had been taken to exercise the paralyzed limb by electricity.

In the second experiment, in addition to dividing the spinal cord below the insertion of the brachial

nerves, as in the first experiment, the principal nerve of *one* of the hind limbs is divided high up near the spine. Two hours afterward, both hind limbs are separated from the body, and their irritability compared by pinching and electrifying the nerves. This is the experiment: the result is, that the "irritability is augmented" in both limbs, but especially in that which had been previously cut off from the influence of the spinal cord by dividing its nerve.

Here, then, are two experiments interesting individually, most interesting in connection. In the first, certain muscles are seen to contract with greater power when they are cut off from the influence of the brain and medulla oblongata. In the second, certain muscles are seen to be more apt to enter into a state of contraction after they are cut off from the influence of the spinal cord than they were before they were so cut off. The facts are altogether unintelligible if the power of muscular contraction is in any way imparted to the muscles by the action of the great nerve-centers. The facts are *not* altogether unintelligible if this power be inveresly related to the amount of nervous influence supplied to the muscles by these centers. At present, however, all that is necessary is to bear in mind the facts as facts, and to leave them to tell heir own story without further comment.

¶ 44. *There is reason to believe that "augmented irritability" does not imply a state of augmented vitality in nerve or muscle.*

Some recent experiments by Dr. Harley* appear

* "Lancet," 7th and 14th June, and 12th July, 1856.

to furnish very good ground for doubting the current belief that strychnia and brucia favor muscular contraction by augmenting the vitality of some vital property of irritability in nerve or muscle.

One of these experiments (one must serve as an example of the rest) consists in removing the hearts of two frogs, and in placing one in a vessel containing simple water, and the other in a vessel containing a very weak solution of strychnia or brucia. This experiment is very simple. The result is one which shows very plainly that the action of strychnia or brucia is not to exalt, but to extinguish the vital properties of the cardiac muscles and nerves—which would seem to contradict altogether the current notion that the spasms arising from the action of these poisons must be looked upon as signs of exalted vitality in some vital property of irritability; for this result is simply this,—that the heart which is immersed in plain water is found to go on beating regularly for some time after the heart immersed in the solution of strychnia or brucia has passed into the state of rigor mortis. And if this be so,—if the "augmented irritability" arising from the action of strychnia or brucia be not connected with a state of augmented vitality in nerve or muscle, it is difficult to imagine any other case of "augmented irritability" in which there can be any such connection.

¶ 45. *It is difficult to believe that "nervous influence" produces the state of contraction in muscle by playing the part of a stimulus to a vital property of contractility in muscle.*

Arguing from the evidence adduced in the pre-

ceding paragraphs, this difficulty would seem to be well-nigh insuperable; and, so far as I know, this evidence is in no sense one-sided.

¶ 46. *It is not difficult to believe that "nervous influence" may play its part in the process of muscular motion through the instrumentality of the natural electricity of the nervous system.*

The natural electricity of the nervous system is an important — perhaps the most important, certainly the most intelligible—element in the composition of "nervous influence;" and, therefore, it is quite possible that the nerves may act upon the muscles by means of their electricity. And certainly there is nothing in the evidence which has been advanced hitherto to make this view in any degree improbable. This evidence has gone to show that the nerves are charged with electricity during the state of rest, and that the state of action in nerves is associated with an electrical discharge analogous to that of the torpedo. This evidence has also gone to show that ordinary muscular contraction is associated with the subtraction of "nervous influence" from the muscles rather than with the addition of such influence to the muscles. It would seem, in fact, as if the *modus operandi* of "nervous influence" in the process of muscular motion were more in accordance with what has been seen to be the *modus operandi* of the electricity of the nervous system in this process, than with the current hypothesis of nervous action; and therefore it may fairly be assumed that the nerves *may* act upon the muscles by means of their electricity. Moreover, it is scarcely to be supposed that

"nervous influence" can act in any way except one which harmonizes with that in which the natural electricity of the nervous system is found to act.

¶ 47. *There is reason to believe that there may be in nervous action a reversal in the electrical relations of the exterior and interior of the nerve-fibers in some parts of the nervous system, and that nervous action may be the consequence of such reversal.*

It has been seen that the natural electrical condition of living nerve is one in which the exterior of the fibers is positive, and the interior negative (¶ 1). It has also been seen that, under certain circumstances, the electrical relations of the exterior and interior of the nerve-fibers may become reversed,—the exterior becoming negative, and the interior positive (¶ 3).

Now, if I have drawn sound conclusions respecting the electrical condition of living nerve during the state of rest, this possibility of reversal in the electrical relations of the exterior and interior of the fiber is a fact of the highest significance—a fact which may be the means of showing how the state of rest in nerve is connected with the state of action. For what are these conclusions? And what is implied in the reversal in question?

I came to the conclusion that the molecules of the *exterior* of the nerve-fiber during the state of rest must be in a state of mutual repulsion, because they are *all* electrified positively; I came to the conclusion that the molecules of the *interior* of the nerve-fiber during the state of rest must be in a state of mutual repulsion, because they are *all* electrified negatively;

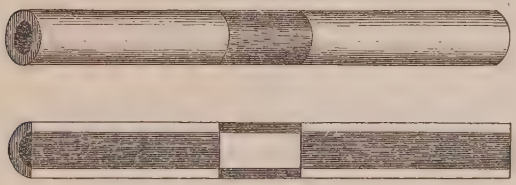
and I did so with good reason, for it is a law of electricity that bodies electrified with similar electricity repel each other. I came to the conclusion, in fact, that the electricity of living nerve during the state of rest is in the condition of statical tension, and not in the current condition; for I was obliged to believe in the existence of some particular constitution of the nerve which kept the exterior and interior of the fibers in opposite electrical conditions—which prevented, that is to say, the dissimilar electricities of the molecules of the exterior and interior of the fibers from yielding to the attraction which tends continually to bring them together, and to produce the electrical discharge by so doing. This was the conclusion at which I arrived with respect to the electrical condition of nerve during the state of rest.

Nor can I see that a different conclusion is necessary in the case where the electrical relations of the exterior and interior of the nerve are reversed, provided only this reversal be general. This is evident; for what is necessary to keep the molecules of the exterior and interior of the fibers in the state of statical tension, is that *all* the molecules of the exterior shall be electrified with *one* kind of electricity, and that *all* the molecules of the interior should be in the same case. So far, indeed, as this result is concerned, it is quite immaterial whether the electricity charging the exterior or interior separately be positive or negative.

But the case is altogether different if the reversal be partial, instead of general. For if the reversal be only partial, it must put an end to the state of statical tension in the exterior and interior of the fiber

by bringing dissimilar electricities together in the exterior and in the interior of the fiber. It must, in fact, substitute a condition of electrical discharge for the previous condition of electrical tension: for to bring dissimilar electricities together, is to leave them free to unite and produce the discharge. All this may, perhaps, be realized more readily by a diagram in which, after the plan adopted previously,

Fig. 22.



the positive parts of the nerve-fiber are shaded lightly and the negative parts darkly: for a moment's glance at this diagram will suffice to show that, in consequence of this reversal in the natural electrical relations of the nerve-fiber (which reversal is represented in the diagram as having taken place at the center of the fiber), the exterior of the nerve will lose its uniform positive character, and become partly positive and partly negative, and that at the same time, and for the same reason, the interior of the nerve will lose its uniform negative character, and become partly negative and partly positive—will suffice to show, in fact, that so long as this state of reversal continues, there can be no state of statical tension in the exterior or interior of the nerve, for all this while positive and negative electricities are so mixed together in the exterior and in the interior as to necessitate the state of electrical discharge. In a word, the

effect of this partial reversal in the electrical relations of the exterior and interior of the nerve-fiber must be to substitute the state of action for the previous state of rest; for, electrically considered, all that is necessary to produce the state of action is a change in which the nerve can furnish a succession of electrical discharges analogous to those of the torpedo.

Hence there is no difficulty in believing that a partial reversal in the electrical relations of the exterior and interior of the nerve-fibers in some part of the nervous system may bring with it, as a necessary consequence, a state of nervous action.

¶ 48. *When a muscle is made to contract by means of its nerve, there is reason to believe that the electrical discharge (analogous to that of the torpedo) which accompanies nervous action has reversed the electrical relations of the exterior and interior of the muscular fibers in the part acted upon; that this reversal has led to the discharge of the electricity which is present in the muscular fibers during the time of rest, and which keeps these fibers in a state of relaxation so long as it is present; and that this discharge of electricity brings about muscular contraction by leaving the muscle free to yield to the action of the attractive force which is inherent in the physical constitution of the muscular molecules.*

It would seem that the phenomena of "induced contractions" (¶¶ 10, 11) are calculated to shed great light upon the way in which the nerves bring about muscular contraction under ordinary circumstances. It would seem that these phenomena, instead of being anything peculiar, are only experiments which

exhibit out of the body what is continually taking place within the body when a muscle is thrown into a state of action by its nerve, or, contrariwise, a nerve by its muscle. For what do these phenomena show? They show that the state of action in nerve-fiber and in muscular fiber is accompanied by an electrical discharge analogous to that of the torpedo. They show that this discharge extends beyond the actual limits of the fiber, and that the "induced" action is the result of the nerve-fiber, or muscular fiber, in which the "induced" action is manifested, being included within the circuit of this discharge. They give a definite view of nervous action, and also of muscular action, which is to be gained in no other way; for they show that this action implies, as it were, the existence of an electrical storm in the atmosphere surrounding the acting fiber, and that the muscular contraction brought about by means of the nerves may arise from the muscular fibers happening to lie within range of this storm. And, certainly, it is quite in accordance with other facts that the electrical discharge accompanying nervous action should tell beyond the actual limits of the nerve. Thus, for example, frogs suspended by silk strings at a distance from an electrical machine will contract when a spark is drawn from the conductor.

Nor is it difficult to go a step further and obtain some precise knowledge respecting the way in which muscular contraction is brought about by the electrical storm which has been mentioned.

M. Chauveau, of Lyons, as has been already seen (¶ 16), is disposed to refer the muscular contraction arising from the action of electricity to the me-

chanical shock attending the passage of the instantaneous current of high tension. He is disposed to refer the electrical and the mechanical causes of muscular action to one and the same category; and in this opinion I fully coincide. On the other hand, it has been seen (¶ 3) that Prof. Du Bois-Reymond has furnished conclusive evidence to the effect that the electrical relations of the exterior and interior of the nerve-fiber and muscular fiber may be reversed by mechanical impressions of various sorts, such as pinching, pricking, and so on. On one side and on the other, there is, indeed, sufficient reason for concluding that the electrical discharge which accompanies the state of action in a motor nerve may *reverse* the electrical relations of the exterior and interior of the muscular fiber in the part of the fiber which happens to come within the range of the discharge. Nay, such an inference is inevitable, if it be true, as it undoubtedly is, that the electrical law of the muscular fiber is precisely the same as the electrical law of the nerve-fiber. And if this be so—if the electrical discharge, analogous to that of the torpedo, which is developed when a motor nerve passes into the state of action, can cause a reversal in the electrical relations of the exterior and interior of the muscular fibers in the parts which happen to lie within range of the discharge—it is to be supposed that the electricity of the muscular fiber will pass from the state of statical tension to that of discharge, as the ulterior result. For as the electrical law of the muscle is in all respects the same as the electrical law of the nerve, it of necessity follows that discharge of electricity must be the result of the reversal in question.

How the change from the state of relaxation to that of contraction is actually produced in the muscular fiber is a question which cannot be fully answered at present. Looking at the question from an electrical point of view, however, it would seem: that the state of relaxation in living muscle is associated with the presence of the natural electricity of the muscle;—that the state of contraction is associated with the absence of this electricity;—that the presence of electricity produces the state of relaxation in living muscle by counteracting the attractive force which is inherent in the physical constitution of the muscular molecules;—and that the absence of this electricity produces the state of contraction in muscle by allowing the attractive force which is inherent in the physical constitution of the muscular molecules to come into play. Viewed in this manner, indeed, it is easy to see that discharge of the electricity belonging to the muscular fibers, however brought about, must, as a matter of course, bring with it muscular contraction.

Nor is it difficult to explain, in accordance with this view, the differences between ordinary muscular contraction and rigor mortis. Ordinary muscular contraction is sudden and transitory. It is sudden, because it is dependent upon the sudden electrical discharge which marks the state of action in the motor nerve: it is transitory, because the discharge producing it is transitory, and because living muscular fibers, when left to themselves, immediately recover the static electrical condition which belongs to the state of rest, and which keeps them in the state of relaxation so long as it continues. Rigor

mortis, on the other hand, is tardy in appearing and more tardy still in departing. It is tardy in appearing, because the natural electricity of the muscles is slow in taking its departure; it continues until the muscular fibers break up in the process of putrefaction, because the natural electricity which produced the state of relaxation in living muscle is no longer present, and because all that is necessary to the continuance of the contraction is the action of the attractive force which is inherent in the physical constitution of the muscular molecules. This is all.

This, no doubt, is a very matter-of-fact view of the process of nervous action in muscular motion, but it is a view which seems to have the effect of simplifying the matter not a little. For what does it do?

It makes the action of the nerves in producing muscular contraction a more intelligible phenomenon by associating an electrical discharge, analogous to that of the torpedo, with the idea of nervous action.

It makes the response of the muscles to the nerves a more intelligible phenomenon, for according to it the electrical discharge which accompanies the action of a motor nerve will have the effect of putting an end to the state of muscular relaxation, by banishing for the moment the natural electricity of the muscle, and of bringing on the state of muscular contraction by thus leaving the muscular fibers free to yield to the action of the attractive force which is inherent in the physical construction of the muscular molecules.

It explains why an action upon any part of a

motor nerve is capable of telling upon a distant muscle: for it is to be supposed that the nerve is an electrical conductor so far as this—that a disturbance in its electrical equilibrium anywhere must affect the nerve everywhere.

It arises, naturally and necessarily, out of the electrical history of nerve and muscle.

It brings the action of nerves upon muscle into the domain of physical and intelligible processes.

But, it may be asked, ought not the state of action in nerve to be accompanied by contraction if this view be true? This question is one which has not been mooted as yet, and I confess that I have not the experimental aptitude necessary to obtain the proper answer to it. At the same time, I may say that it is by no means certain that there is no contraction in nerve-fiber under these circumstances, and I may even point to the beaded appearance in the nerve-fibers after death as a ground—somewhat unstable it is true—for believing that the state of action in nerve-fibers may involve the state of contraction in those fibers. For, looking at this fact, I cannot but think that these beads could not have been formed unless there had been a longitudinal contraction toward certain points in the nerve-fibers —a contraction by which a substance in the fibers, which was before uniformly continuous, has become broken up between these points. I cannot but think that these beads may show that there is, contemporaneously with the establishment of rigor mortis, a change in the nerve-fibers which is strictly parallel to that which takes place at that time in the muscular fibers—a change in which there is decrease of

length with a comparative increase of breadth; for if the presence of the beads implies contraction in the longitudinal direction of the nerve-fibers, it is not difficult to believe that it may imply also bulging out in the lateral direction. And if this be so, then I may fairly go a step further, and assume that there is a corresponding change in nerve-fiber during nervous action: for if the change in muscular fiber in ordinary muscular contraction and in rigor mortis is one and the same, there is, after what has been said, some ground for believing that the physical change in nerve-fiber after death is not remotely akin to the change which passes over the nerve-fiber in nervous action,—that, in fact, the nerve in acting passes into a state of contraction, instead of remaining motionless.

Be this as it may, however, enough has been said to justify the statement which stands at the head of the present paragraph; and this is all that is necessary now.

¶ 49. *There is reason to believe that the general convulsion arising from hæmorrhage and suffocation —which phenomena were left unexplained in the last section—may be explained by supposing that the electrical relations of the exterior and interior of the nerve-fibers in the medulla oblongata are reversed when the supply of arterial blood to this center falls below a certain amount, and that the nerves of the whole muscular system are thrown into a state of action in consequence of this reversal.*

In general convulsion, there are reasons (which I

must not stay to specify) which make it certain that the medulla oblongata is the nerve-center especially affected; and therefore—still looking at the matter from the previous point of view—the question for present consideration concerns the probable effect of loss of blood upon the electrical condition of this center. Is it possible that this loss may have given rise to a reversal in the electrical relations of the exterior and interior of the nerve-fibers of this organ? Such a supposition is certainly not impossible; such a supposition can scarcely be regarded as improbable: for as there is such a reversal in the spinal cord of frogs before the final extinction of the vitality of this nerve-center (¶ 3) there is sufficient ground for believing that the electrical relations of the exterior and interior of the fibers of the medulla oblongata may be reversed in like manner when the want of arterial blood has caused the vitality of this center to sink below a certain point. And if this be so, then there must not only be discharge of electricity between the part of the nervous system in which the electrical relations of the exterior and interior of the nerve-fibers are reversed, and those parts in which these relations are not so reversed, but there must be general convulsion also; for the part of the nervous system which is especially implicated in the discharge is that which is, in a peculiar sense, at the very center of the muscular system. Nor is it difficult to see why, after a certain time, this convulsion may come to an end. For, after a certain time, it is conceivable that the discharge may fail in the force necessary to produce this result. Or else it may be supposed that the

loss of blood eventually tells upon other nerve-centers and nerves, producing a reversal of the electrical relations of the exterior and interior of the nerve-fibers in them also, and in this way putting an end to the conditions of the discharge: for when all parts of the nervous system are in the same state of reversal, the whole system must return (¶ 47) to that condition of the statical equilibrium which is characteristic of the state of rest as contradistinguished from the state of action.

Nor is there any essential difference between the antecedents of convulsion from hæmorrhage and the antecedents of convulsion from suffocation. In the one case, the vitality of the medulla oblongata fails as the blood streams away, and at a certain point in this process of failure the electrical relations of the exterior and interior of the fibers of this center become reversed: in the other case, the same results are brought about at a given moment by stoppage of the respiration, and by the deficiency of arterial blood arising therefrom. In each case, it is failure of vitality from want of arterial blood which leads to the reversal which brings about the convulsion, and nothing else. According to this view, indeed, there is no room for the notion that venous blood has a special power of producing convulsion by acting as a stimulus to a vital property of irritability belonging to the medulla oblongata. Nay, if this view be true, there is no room left for that current theory which seeks to explain muscular contraction by supposing that a vital property of irritability in nerve and muscle has been stimulated into action.

¶ 50. *There is reason to believe that nervous action arising from a local cause, mechanical or other, may be explained by supposing that this cause of action has produced a local reversal in the electrical relations of the exterior and interior of the nerve-fiber, and that this reversal produces the state of nervous action.*

It has been seen that the natural electrical relations of the exterior and interior of the nerve-fibers may be reversed by various causes, mechanical and others, without altering materially the vital properties of the nerve (¶ 3); and therefore it is a fair deduction from what has just been said, that the local cause which produces action in a motor nerve is attended by such reversal. And if so, then a state of change must be set up which may, in the manner already explained, cause the nerve to pass from the state of rest into that of action. Moreover, with such an intercommunion as there is between the different parts of the nervous system, it is plain that this state of action, once set up, may extend to distant parts, and give rise to convulsion, spasm, and other muscular disturbances. In the case of convulsion, for example, it is not difficult to imagine that this state of action may be propagated along the vaso-motor nerves to the vessels of the medulla oblongata, that contraction in these vessels may be the consequence, and that the diminished supply of blood arising from this cause may do precisely what is done at a given point in the progress of hæmorrhage or suffocation,—that is, lead to a state of general convulsion by reversing the electrical relations of the exterior and interior of the nerve-fibers

in this center. And in the case of spasm, it is equally possible that an analogous result may be brought about by a similar action having been transmitted through the vaso-motor nerves to the vessels of certain not very clearly defined parts in the optic thalami, corpora quadrigemina, crura cerebri, pons Varolii, crura cerebelli, medulla oblongata, or upper part of the spinal cord, one or all.

¶ 51. *There is reason to believe that the state in nerve which is known under the name of "irritation" may be explained by supposing that the electrical relations of the interior and exterior of the nerve-fibers are reversed in some part of the nervous system, and that this reversal may keep the nervous system, locally or generally, in the necessary state of action.*

This reason is to be found in the contents of the paragraphs immediately preceding the present one; and if moderate attention have been paid to what has been said, there can be no difficulty in finding it. If, indeed, there be a state of reversal in the electrical relations of the exterior and interior of the nerve-fiber in any part of the nervous system, it follows from the premises that there must be a state of nervous action: and this state of nervous action, in one form or other, and in one part or other, is all that is wanted to account for everything which is included in the idea of "irritation." Moreover, this view is not quite so incomprehensible as that which is currently accepted as more or less satisfactory.

¶ 52. *There is no difficulty in believing that the will may avail itself of the aid of the natural electricity*

of the nervous system in the case of voluntary muscular action.

Natural electricity disappears in every act of voluntary muscular contraction. Of this fact M. Du Bois-Reymond has supplied abundant proof. It is also possible to obtain "induced contractions" (¶¶ 10, 11) from contractions which are produced voluntarily. This I have done on several occasions. In voluntary muscular contraction, indeed, there is the same evidence of electrical discharge as that which is found to accompany other forms of muscular contraction; and, therefore, after what has been said, there is reason enough for the assumption that the will may avail itself of the help of the natural electricity of the nervous system in the production of muscular motion. And, so far as I can see, there is nothing in this view which in any sense compromises the dignity of the will as the paramount expression of vital power.

¶ 53. *There is reason to believe that "nervous influence," apart from nerve-electricity, is, to say the least, a very indefinite idea.*

The previous considerations respecting the action of "nervous influence" in the process of muscular motion lead me to this conclusion, and I cannot see what other conclusion is open to me. At the same time, I fully believe that "the electric chain wherewith we are darkly bound" has its ends more firmly fixed in the invisible world than in the visible. In other words, I fully believe that what is called electricity, is only a one-sided aspect of a law which, when fully revealed, will be found to rule over vital

as well as physical phenomena—not materializing the former, but spiritualizing the latter, and ennobling both: for the law which is sufficient for both purposes is more universal, and therefore more spiritual and noble, than that which is only sufficient for one purpose.

(3) *ON THE PART WHICH CERTAIN COMPARATIVELY UNIMPORTANT NON-ELECTRICAL AGENTS HAVE TO PLAY IN THE PROCESS OF MUSCULAR MOTION.*

¶ 54. *When a motor nerve or muscle is thrown into a state of action by mechanical or chemical causes, or by heat, or cold, or light, there is reason to believe that the electrical relations of the exterior and interior of the nerve are reversed in the part acted upon, and that the state of action may be the result of this reversal.*

That this may be the case with respect to a motor nerve has been already seen (¶ 49). Nor is the case different when the state of action in a motor nerve is a reflex phenomenon: for here all that is necessary is to suppose that the cause producing action, by reversing the electrical relations of the exterior and interior of the nerve-fibers in the part acted upon, is removed a step further back—is removed from the efferent to the afferent portion of the nervous arc. And so, likewise, when the muscle is thrown into a state of contraction by acting upon the muscular fiber itself; for, as the electrical law of muscle and nerve is one and the same, it may be supposed that the agent producing contraction has caused a local reversal in the electrical relations of the exterior and interior of the muscular fiber, and that this reversal

causes the fiber to pass from the state of rest into that of action in the manner already explained.

It would be easy to support these general conclusions by reference to particular cases; but it is not necessary. And well it is that it is not necessary; for the position of the finger on the dial of the time-piece warns me that I ought to have brought these remarks to a close some time ago.

LECTURE IV.

In the last Lecture I investigated at some length the *modus operandi* of the blood, and of "nervous influence," so called, in the process of muscular motion, and I came to a conclusion which is altogether adverse to the current theory on the subject. I came, indeed, to the conclusion that these agents bring about the state of muscular contraction, not by acting as *stimuli* to a vital property of irritability in nerve and muscle, but by altering in a definite and intelligible manner the electrical condition of the nerve and muscle.

In the present Lecture I have more to do than I can hope to do well in the time at my disposal. I have to say something about the theory of ordinary muscular motion which appears to be necessitated by the evidence advanced hitherto. I have to say something upon the physiology of rhythmical muscular motion. I have also to say something upon the physiology of sensation.

§ IV. On the theory of muscular motion which appears to arise out of the premises.

¶ 55. *The true theory of muscular motion appears to be a purely physical theory.*

The primary deductions from the evidence advanced hitherto appear to be these:—

That there are unmistakable signs of natural electricity in living nerve and muscle during the state of rest.

That the natural electricity which is present in living nerve and muscle during the state of rest is in the statical and not in the current condition.

That living muscle, when left to itself, is kept in the state of relaxation by the statical action of its natural electricity.

That an electrical discharge analogous to that of the torpedo is developed in the neighborhood of nerve or muscle during the state of action.

That a nerve or muscle is for the moment deprived of its natural electricity whenever it is thrown into the state of action by the shock of a coil-machine, or by any other artificial means.

That the action of a motor nerve in producing muscular contraction is one which may deprive the muscle of its natural electricity; for it may be supposed that the muscular fibers lie near enough to the nerve-fibers to be within the range of the electrical discharge (analogous to that of the torpedo) which is developed in the neighborhood of the nerve during the time of nervous action, and that the muscular fibers are deprived of their natural electricity by the shock of this discharge, in precisely the same way as that in which they are so deprived by the shock of the current of the coil-machine.

That muscle deprived of its natural electricity passes into the state of contraction, because muscle so deprived is left free to yield to the

action of the attractive force which is inherent in the physical constitution of the muscular molecules.

That ordinary muscular contraction is *not* continuous, because the electrical condition of living muscle is such that immediately after the state of contraction is brought about by the loss of electricity, the opposite state of relaxation is restored by the recovery of electricity.

That muscle and nerve have ceased to exhibit any sign of natural electricity before the occurrence of rigor mortis.

That the contraction of rigor mortis is continuous, because the attractive force which is inherent in the physical constitution of the muscular molecules is then no longer antagonized by the natural electricity of the muscles and nerves.

That ordinary muscular contraction and rigor mortis are only different aspects of the same process.

That there is no ground for believing that a vital property of irritability has to do with the action of a motor nerve, or that a vital property of irritability or tonicity is concerned in bringing muscle into the state of contraction.

As it seems to me, indeed, the whole tenor of the previous evidence goes to show that the true theory of ordinary muscular motion is a purely physical theory; and I, therefore, adopt this theory provisionally, and proceed, without further comment, to inquire whether it is confirmed or contradicted by the evidence which yet remains to be dealt with.

¶ 56. *This theory of muscular motion is not contradicted by any facts which render it necessary to suppose that the natural electricity of the muscular system is converted into contractile force at the time of contraction.*

The doctrine of the "correlation of the physical forces" has suggested the notion that the natural electricity of the muscular system may be converted into contractile force, and that muscular contraction may be the consequence of such conversion. But this view, as it seems to me, is not in harmony with the history of rigor mortis. For what does this history show? It shows, in brief:—(1) that the natural electricity of the body disappears slowly after death; (2) that rigor mortis is deferred until this disappearance is complete and final; and (3) that, once established, it remains without any intervals of relaxation until the muscular fibers break up in the ruin of putrefaction. The case is *not* one in which the natural electricity fails suddenly, and as suddenly gives place to contraction, and in which, therefore, it might be supposed that the electricity has been transformed into contractile force: it is one in which, after failing gradually, the electricity has become reduced to so very insignificant an amount before the time for rigor mortis has arrived, as to make it difficult to know where to find the electricity which is supposed to be transformed into contractile force when the contractile force is wanted. The case, too, is one in which the very duration of the contraction would seem to be sufficient to show, in no obscure manner, that the contraction is connected with the physical integrity of the muscular

fibers, and with nothing else: for the simple fact is, that rigor mortis, once established, is only put an end to by the putrefactive decomposition of the contracted fibers. In a word, the history of rigor mortis is as intelligible upon the view which refers this form of contraction to the attractive force which is inherent in the physical molecules of the muscular fibers, as it is unintelligible upon the view which would connect it with a contractile force arising in the transformation of animal electricity. And if this be so with respect to rigor mortis, it is fair to assume that it will be so also with respect to ordinary muscular contraction: for the explanation which will apply to ordinary muscular contraction as well as to rigor mortis, is surely to be preferred to that explanation which at best can only apply to ordinary muscular contraction.

¶ 57. *This theory of muscular motion is not contradicted by any peculiarity in the law of muscular contraction which does not admit of a physical explanation.*

No difficulty can be raised upon this point, for it is now generally admitted that the law of muscular contraction differs in no respect from the law of contraction in India-rubber and other elastic bodies.

¶ 58. *This theory of muscular motion is not contradicted by the fact that dead muscle is less strong and tough than living muscle, for this phenomenon admits of a physical explanation.*

After death, it is easy to believe that the muscular fibers may be weakened by the solvent action of

the fluid analogous to gastric juice—the "juice of flesh"—which is contained in muscular tissue, or by the commencing resolution of the muscular molecules into their constituent elements. After death, it is also easy to believe that the strain upon the muscular fibers may fail to produce that state of contraction in the fibers which it would not fail to produce during life, and that, for this reason, dead muscle may be less strong and tough than living muscle: for it is to be supposed that the muscular attraction in the muscular fibers will oppose a greater resistance to this strain when the molecules approximate, as they do in a state of contraction, than that which is opposed when the molecules are apart, as they are in the state of relaxation. But, be the explanation what it may, enough has been said to make it altogether gratuitous to suppose that dead muscle is less tough and strong than living muscle, because death has destroyed a vital property of contractility.

¶ 59. *This theory of muscular motion is not contradicted by the fact that muscle cannot act without intervals of rest, for this phenomenon admits of a physical explanation.*

That muscular action is dependent upon certain *physical* conditions which become deranged by the continuance of this action, and which cannot again be set in order until nutrition has had time to do its work, is readily conceivable: and surely this view is quite as probable and quite as intelligible as that which would account for these phenomena by supposing that a vital property of irritability is after a

time tired out by overwork, and that rest is necessary for the revivification of this property.

¶ 60. *This theory of muscular motion is not contradicted by the fact that muscle may contract and relax without change of volume and without loss of time, for these changes have their strict parallels among purely physical phenomena.*

There are certain experiments by Mr. Joule, of Manchester,* which show that a bar of iron, suddenly and without any change of volume, gains in length and loses in breadth when it is charged with magnetism, and that it as suddenly returns to its former dimensions when the magnetism is discharged.

In one experiment, a square bar of iron, with one of its ends fixed, and with the other end in communication with a system of levers by which any change in its length is multiplied 3,000 times, is placed in the longitudinal axis of a coil composed of insulated copper wire, and after this, it is alternately magnetized and demagnetized by alternately making or breaking the connection between the coil and a Daniell's battery of half a dozen cells. This is what is done; what happens is this—that when the bar becomes charged with magnetism, the needle of a dial, which records the movements of the system of levers connected with the end of the bar, immediately springs forward to the extent of a quarter of an inch or thereabouts—a movement which shows that the effect of the charge of magnetism has been to cause the bar to gain in length to

* "Philosophical Magazine," February and April, 1847.

the extent of $\frac{1}{3000}$th of an inch; and that when the bar loses its charge of magnetism, the needle immediately springs back to the position it occupied before receiving the charge. And in addition to these sudden forward and backward movements of the needle—the movements obviously arising from the charge and discharge of magnetism—there is also a slow forward movement if the coil be kept connected with the battery—a slow movement arising, as it would seem, from the expansion of the bar under the action of the heat radiating from the current in the coil: but this slow movement is quite distinct from the sudden movements, and that it is so is proved by the fact that the shifting of the finger on the dial to another position under the slow movement does not alter the amount of forward or backward motion which is connected with the charge and discharge of magnetism.

In this experiment, it is seen that a bar of iron suddenly gains in length when it is charged with magnetism, and as suddenly loses its length when the magnetism is discharged: in the experiment which has next to be noticed, it is seen that these changes are unaccompanied by any alteration in volume—that, in fact, the gain in length is accompanied by a compensative loss in breadth.

This companion experiment is as follows:—

A conductor consisting of ten insulated copper wires, each wire being $\frac{1}{20}$th of an inch in diameter and 110 yards in length, is coiled around a glass tube, forty inches in length and one inch and a half in diameter. One end of this tube is closed permanently in glass; the other end has a cork pro

vided with a vent-hole and having a graduated capillary tube fitted into this hole and projecting from it. The graduation of the capillary tube is made upon a scale of which one degree is equal to $\frac{1}{450000}$th part of the bar which has to be magnetized and demagnetized. The bar itself is of annealed iron, one yard in length and half an inch in diameter. In proceeding with the experiment, this bar is placed in the tube within the coil; then water is poured in so as to fill this tube; then the cork is adjusted so as to force the water to a convenient height in the capillary tube projecting from the vent-hole in the cork; and finally, the bar is alternately magnetized and demagnetized by alternately connecting and disconnecting the coil with a Daniell's battery of half a dozen elements. This is the experiment; the result is this—that the level of the fluid in the capillary tube is not affected by the connection or disconnection of the coil with the battery. The result, that is to say, is one which shows very plainly that the alternate charge and discharge of magnetism has produced no alteration in the volume of the bar; for if it had been otherwise, the sudden changes in the length of the bar (of which there was evidence in the last experiment) would cause the level of the fluid in the capillary tube to rise twenty degrees when the bar is charged with magnetism, and to fall as many degrees when this magnetism is discharged. In other words, this experiment shows very plainly that the increase of length which the bar undergoes when charged with magnetism, and of which there was evidence in the last experiment, is accompanied by a compensative

decrease of breadth—that, in fact, the changes in form are not accompanied by changes in volume.

In this last experiment, the level of the water in the capillary tube is found to rise slowly if the coil be kept in connection with the battery, and this slow movement is evidently owing to the same cause as that which produced the slow movement of the finger upon the dial in the first experiment, namely, the expansion of the magnetized bar under the action of the heat given out by the galvanic current in the coil. At any rate, it is evident that this slow rising in the level of the water in the capillary tube, under these circumstances, does not invalidate the fact that the level of this water does not undergo any alteration at the moment when the bar is charged with magnetism by connecting the coil with the battery, or at the moment when this magnetism is discharged by breaking this connection: and this is the fact which is of interest in the present inquiry.

It is plain, then, that a bar of iron may suddenly and without any change of volume gain in length and lose in breadth when it is charged with magnetism, and that it may as suddenly return to its former dimensions when this magnetism is discharged,—it is plain, that is to say, that a bar of iron, under these circumstances, may undergo changes which are strictly parallel to the changes of muscular fiber which constitute the opposite states of contraction and relaxation; and, therefore, it is fair to conclude that these changes in muscular fiber are not inconsistent with the physical theory of muscular motion which is now under consideration.

¶ 61. *This theory of muscular motion is not contradicted by any fact which renders it necessary to suppose that an increased disposition to muscular contraction is connected with a state of exalted functional activity in a vital property of irritability in motor nerve or muscle.*

In support of this statement, it is only necessary to refer to the facts which contradict the notion (¶ 44) that the spasms of poisoning by strychnia and brucia are to be looked upon as signs of exalted vitality in the vital property of irritability with which the nerves and muscles are endowed by common consent. For if it be necessary to come to this conclusion with respect to these spasms, it is difficult to imagine any other case in which a different conclusion can be arrived at.

¶ 62. *On the other hand, this theory of muscular motion would seem to derive no small amount of confirmation from the fact that it explains muscular relaxation no less than muscular contraction; that it is equally applicable to ordinary muscular contraction and to rigor mortis; and that it brings the phenomena of muscular motion into subjection to physical law.*

The current theory of muscular motion makes no account of the state of muscular relaxation, and it is obliged to assume the existence of two different vital properties of contractility—irritability and tonicity—in order to explain the differences between ordinary muscular contraction and rigor mortis. Hence the current theory fails in comprehensiveness when it is compared with the theory under consideration. And

for the rest, I will only say this—that a theory based upon the known laws of physics may have some claim to be regarded as supplying a satisfactory explanation; but that a theory which assumes as its fundamental principle that the mystery of life is a sufficient explanation for every difficulty can have no such claim. For what explanation is it to say that a nerve produces contraction in muscle, or that a muscle contracts, because muscle and nerve are endowed with a living capability of doing so?

B. ON RHYTHMICAL MUSCULAR MOTION.

Under this heading all I can do is to glance at the beating of the heart, the peristaltic movements of the alimentary canal, and the respiratory movements of the chest. I omit much, but, so far as I know, I omit nothing which does not readily harmonize with the facts which have to be cited and with the conclusions which have to be drawn.

§ I. On the beating of the heart.

¶ 63. *The blood would seem to play one and the same part in the movements of the ventricles and in ordinary muscular motion.*

The passage of the ventricles of the heart from the state of systole into that of diastole is contemporaneous with a rush of red blood through the coronary arteries into the walls of the heart. The return of the ventricles of the heart from the state of diastole into that of systole is contemporaneous with a pause in which fresh blood is not injected into the walls of the heart through the coronary vessels, and in which, moreover, sufficient time is allowed for the

blood injected previously to have acquired venous properties. It would seem, indeed, as if the blood has to play one and the same part in the movements of the ventricles and in ordinary muscular motion: for it has been seen that the presence of arterial blood is favorable to the restoration and preservation of that state of static electrical equilibrium which produces muscular relaxation, and that the absence of arterial blood gives rise to that disturbance in this electrical equilibrium which produces temporary muscular contraction.

¶ 64. *"Nervous influence" would seem to play one and the same part in the movements of the ventricles of the heart and in ordinary muscular motion.*

Mr. Paget* has proved in a very satisfactory manner that the rhythmical movements of the heart are ruled by certain nerve-centers which have been detected by MM. Bidder† and Rosenberger‡ in the substance of the heart, and which are found clustered together chiefly in the lines of junction between the auricles and the ventricles, and between the auricles and the great veins. Some of Mr. Paget's experiments show that when the hearts of frogs or tortoises are removed from the body and cut into fragments, it is not every fragment which has the power of going on beating. They show, in fact, that every fragment has, like ordinary muscle, the power of contracting vigorously when pricked, or pinched, or

* "On the Cause of the Rhythmic Motion of the Heart," Proc. of Royal Society, 28th May, 1857.

† Müller's "Archiv," 1852, p. 163.

‡ "De Centris Motuum Cordis." 8vo. Dorport, 1850.

otherwise disturbed; but that the fragments which have the power of beating rhythmically are only those which contain some of those peculiar nerve-centers,—only those which had occupied a position on or near the lines of junction between the auricles and the ventricles, or between the auricles and the insertion of the great veins. And other experiments belonging to the same set show that the effect of crushing these nerve-centers by inclosing the parts in which they are found in tight ligatures is at once to put a stop to rhythmical movement. Thus, for example, rhythmical movement is arrested in the auricles, but not in the ventricle, if a ligature be tied tightly around the heart of a tortoise at the line of junction between the auricles and the great veins entering into them. In a word, what Mr. Paget has attempted to prove by these experiments, that he has succeeded in proving most satisfactorily.

What, then, is the *modus operandi* of these nerve-centers of the heart—these "rhythmic nerve-centres," as Mr. Paget calls them—in the movement of the ventricles? Is it possible that the stoppage of the supply of blood to these nerve-centers, which stoppage takes place when the ventricles pass into the state of diastole (¶ 63), may bring about the state of systole in the ventricles, in the same way as that in which general convulsion is brought about by shutting off the supply of blood to certain nerve-centers within the head? Is it possible that the systole of the ventricles may exhibit on a small scale, and naturally, what is exhibited on a large scale, and artificially, in the experiments of Astley Cooper and MM. Kussmaul and Tenner, to which attention has

been already called (¶ 41)? Such a supposition is not at all impossible; such a supposition is not at all improbable. Indeed, the only key wanting for the interpretation of the rhythm of the ventricles appears to be that which is supplied by the view of nervous action set forth in these Lectures. For what is the case according to this view? The case, in the first place, is this—that the nerve-centers of the heart will be in a state of rest so long as the coronary vessels are supplied with a sufficiency of arterial blood, and that the walls of the ventricles, being thus left to themselves, will be in the state of relaxation which constitutes their diastole. The case, in the second place, is this—that the nerve-centers of the heart will pass into a state of action when they are no longer supplied with a sufficiency of arterial blood, and that this action will bring with it that state of contraction in the walls of the ventricles which constitutes the systole. In this case, that is to say, the stoppage in the supply of blood to the nerve-centers of the heart, which stoppage occurs when the ventricles pass into the state of diastole, will bring with it, sooner or later, a reversal in the electrical relations of the exterior and interior of the nerve-fibers of these centers; and this reversal will lead, in the manner which has been sufficiently explained already (¶¶ 47, 48), to the electrical discharge, analogous to that of the torpedo, which would seem to be the essential element in nervous action. According to this view, indeed, there would seem to be no great difficulty in explaining how the systole of the ventricles may be brought about. And this is the main difficulty

for present consideration; for, after what has been said, the opposite state of diastole is the necessary consequence of that state of relaxation into which muscles naturally fall when the nerves belonging to the muscles are not in a state of action.

¶ 65. *The differences which distinguish the rhythm of the auricles from that of the ventricles, may be resolved, in the main, into simple consequences of the movements of the ventricles.*

The diastole of the auricles, which is virtually coincident with the diastole of the ventricles, is a problem of no great difficulty; for there is reason to believe that this state is due partly to the same cause as the diastole of the ventricles—namely the rush of blood into the coronary vessels, and partly to the current of blood which is continually setting in from the great veins. The systole of the auricles, which is contemporaneous with the diastole of the ventricles is less readily accounted for. Upon examination, however, there is reason to conclude that this state may be partly, if not mainly, due to the falling in of the walls of the auricles upon the blood being suddenly sucked from the auricles into the ventricles: and this reason is to be found in the absence of valves at the mouths of the great veins opening into the auricles. For if the systole of the auricles had to minister to the carrying on of the circulation in the same sense as that in which the systole of the ventricles has to minister—that is, by actively contracting upon the blood,—is it not fair to assume that there would be valves at the mouths of the

great veins to prevent the reflux of blood from the auricles into these vessels?

¶ 66. *The manner in which the movements of the heart are affected when the medulla oblongata or the pneumogastric nerves are subjected to the action of electricity need lead to no different conclusions respecting the action of the heart.*

The action of the heart may be immediately brought to a stand-still by subjecting the medulla oblongata or the pneumogastrics of a frog or dog to the shocks of a coil-machine. The brothers MM. Ernest and Henri Weber* were the first to gain a distinct view of this fact; but before them Professor Claude Bernard† had obtained a glimpse of it, for once, while auscultating the chest of a dog whose pneumogastrics were included in the circuit of a coil-machine, he had noticed that the sounds of the heart were not to be heard so long as the coil was in action. More recently, however, Professor Lister‡ has shown that the movements of the heart are not always arrested under these circumstances. He has shown, indeed, that feeble shocks have a directly contrary action to that of powerful shocks; and the following is one of the experiments by which he has done so. The animal experimented upon is a rabbit; the plan of proceeding is to include the vagus in the circuit of a coil-machine,

* "Handwörterbuch der Physiologie," art. Muskelbewegung, vol. iii. p. 42. 1846.

† Thèse de M. le Dr. Lefèvre. Paris, 1848.

‡ "Preliminary Inquiry into the Functions of the Visceral Nerves, with special reference to the so-called 'Inhibitory System,'" Proc. of Royal Society, 13th August, 1858.

and to vary the strength of the shocks which are made to act upon the nerve. In the first place, the shocks are made doubly feeble by charging the galvanic cell belonging to the coil with very weak acid, and by removing the core of iron wire from the hollow of the coil. In the next place, the shocks are increased in strength by pushing the core of iron wire home within the coil. In the third place, an interval of fifteen or twenty minutes is allowed to elapse, and then the shocks are still further increased in strength by adding a little more acid to the galvanic cell. The result, so far as the action of the heart is concerned, is as follows: quickening of the rhythmical movements in the first stage of the experiment; arrest of these movements in the state of diastole in the second stage; renewal of these movements in the third stage. At first sight the result appears to be unintelligible and contradictory, but it is not so in reality. One thing which must be borne in mind in explaining it is this—that the electrical discharge, analogous to that of the torpedo, which accompanies the action of the pneumogastric nerve, is proportionate to the amount of action in the nerve, and that this amount of action is to be measured by the amount of action in the coil-machine—so long, at least, as the nerve retains a fair measure of its electricity. And another thing not less necessary to be borne in mind is this—that the electric discharges, analogous to those of the torpedo, which accompany the action of the pneumogastric nerve, tell upon the movements of the heart by acting upon the rhythmic nervous centers of the heart. Bearing these things in mind, there would

seem, indeed, to be no great difficulty in explaining what has to be explained. In the first stage of the experiment, when the pneumogastric is acted upon by feeble shocks, all that is necessary to explain the increased rhythmical movements of the heart at this time is to suppose that the electrical discharges which attend the action of the nerve are just of the strength necessary to produce a corresponding state of action in the nerve-centers within the heart. In the second stage of the experiment, when the pneumogastric is acted upon by strong shocks, all that is necessary to account for the cessation of the heart's action in the state of diastole, is to suppose that the electrical discharges which attend upon the action of the nerve are strong enough to paralyze the nerve-centers within the heart: for to paralyze these centers, is to leave the muscles of the heart to themselves, and to leave muscles to themselves, is to leave them in the state of relaxation—a state which, in the case of the heart, implies the state of diastole. And in the third stage of the experiment, when the pneumogastric is exposed to still stronger shocks, it is equally possible to explain the renewal of the heart's action; for at this time it is to be supposed that the natural electricity of the pneumogastric has become greatly diminished, and that, for this reason, the electrical discharge attending the action of the nerve, instead of having a paralyzing power over the nerve-centers within the heart, as was the case in the second stage of the experiment, is only of the strength necessary to bring about the same result as that which was obtained in the first stage of the experiment.

In a word, there is nothing in the facts which have just been under consideration to render it necessary to doubt the conclusions which have been already arrived at respecting the movements of the heart, and muscular motion generally.

¶ 67. *The fact that a heart may go on beating after it is removed from the body, and that certain fragments of a heart may retain this power, need lead to no different conclusions respecting the action of the heart.*

Does the oxygen of the air penetrate into the muscular substance of the heart and act upon the rhythmic nerve-centers in the same manner as that in which the oxygen of the arterial blood has been seen to act? Does the using-up of the oxygen of the air in the "respiration of the tissues" have the same effect upon the rhythmic nerve-centers as that which is seen to arise from the using-up of the oxygen of the blood when the blood passes from the arterial to the venous state? If this be so—and it is difficult to find any reason why it may not be so—it is plain that the rhythmical movements in question may go on as long as the rhythmic nerve-centers preserve their natural electricity; for it may be supposed that the systolic contraction following the using-up of the oxygen of the air will displace the air which has thus been used up, and provide room for the admission of fresh air, and that the oxygen of this fresh air will do what was done by the oxygen in the first instance. And certainly it is an argument in favor of this view, that a heart or a fragment of a heart, which is capable

of beating out of the body, will cease to beat when it is placed in a vacuum, or when it is plunged in an atmosphere of nitrogen, hydrogen, or carbonic acid, and that it will resume its beatings when air is admitted into the vacuum, or substituted for the gases which have been mentioned. Nor is a contradictory conclusion to be drawn from the fact that the heart, or fragment of heart, which has ceased to beat in common air, will begin to beat again, and will go on beating for some time, when it is removed from an atmosphere of common air into one of oxygen gas.

¶ 68 *The manner in which the contractile coats of the vessels are affected by the action of the vaso-motor nerves need lead to no different conclusions respecting the action of the heart and of the muscular system generally.*

A century ago or more, Parfour du Petit* described several of the effects which result from the division of the sympathetic nerve in the neck; but it is to Professor Claude Bernard† and Dr. Brown-Séquard‡ that physiologists are chiefly indebted for a full and exact knowledge of thes effects, and also of those which result from the action of electric shocks upon the peripheral portion of the trunk of

* "Mémoires de l'Académie des Sciences," 1727.

† "Comptes Rendus de la Soc. de Biologie," Déc. 1851; "Gaz. Méd. de Paris," 1852, p. 72; "Comptes Rendus de l'Acad. des Sciences," 28 Mars, 1852; "Leçons sur la Physiologie et la Pathologie du Système Nerveux," Paris, 1858, vol. ii. Leçons 15 et 16.

‡ "Philadelphia Med. Examiner," Aug. 1852; "Exper. Researches applied to Physiology and Pathology," New York, 1853; "Lancet," 30th Oct. 1858.

the divided nerve—to the former physiologist, for pointing out the greater number of the consequences of dividing the nerve; to the latter physiologist, for describing the greater number of the consequences of electrifying the nerve. In these matters, indeed, the names of these two great physiologists will ever be inseparably connected, not only as having discovered facts which are complementary the one to the other, but as having in more than one instance discovered the same fact, and enunciated it almost simultaneously.

The effect of removing the interior cervical ganglion or of dividing the cervical filament of the sympathetic nerve in a rabbit, is rapid and unmistakable increase in the warmth and vascularity of the corresponding side of the head and face—the temperature rising several degrees, the eye, nostril, and ear becoming bloodshot, the pulse acquiring both force and fullness—and this effect may continue with little or no change for weeks, perhaps for months. The effect of exposing the peripheral portion of the trunk of the divided nerve to the shocks of a coil-machine is at once to put an end to the state of increased warmth and vascularity which results from the division of the nerve. Moreover, the latest investigations of Professor Claude Bernard in this subject have shown that the effects of dividing and electrifying the vaso-motor nerves of the limbs is precisely the same as those which have just been described as arising from these operations upon the vaso-motor nerves of the neck: and thus, dilatation of the vessels may be spoken of as a general consequence of paralysis of the vaso-motor nerves, and

contraction of the vessels as a consequence, not less general, of the action of these nerves.

How, then, are these facts to be accounted for? Why do the vessels become dilated when they are paralyzed by dividing their special nerves? How is it that they are kept in a state of contraction before they are paralyzed in this manner?

In order to understand why it is that the vessels should be partially contracted so long as their nerves are uninjured (it is expedient to take this question first) all that appears to be necessary is to suppose that the electrical relations of the exterior and interior of the fibers of these nerves—which relations are preserved in their natural condition so long as these fibers are acted upon by arterial blood—become reversed when this arterial blood is transformed into venous blood,—that this reversal sets up a state of action in the vaso-motor nerves,—and that this state of action in the vaso-motor nerves keeps the muscular coats of the vessels in a state of contraction so long as it lasts. And for the rest, it is only necessary to suppose that the new pulse of arterial blood puts an end for the time to that state of action in the vaso-motor nerves which leads to contraction in the vessels, by restoring the electrical relations of the exterior and interior of the nerve-fibers to the state which existed before the occurrence of that reversal which gave rise to the state of action. All this is the necessary consequence of that view of nervous action which was set forth in the last Lecture (¶¶ 47–53), and upon which, however necessary, any further comment is now impracticable for sheer want of time.

And if this be the state of the case as long as the vaso-motor nerves preserve their proper vital relations to the vessels, it follows that dilatation of the vessels must be the result of dividing the vaso-motor nerves: for when these nerves are divided, the muscular coats of the vessels must be left to themselves; and when these coats are left to themselves they must, as a matter of course, pass from that state of contraction, which depends upon the action of the vaso-motor nerves, into that state of relaxation, which is the natural state of muscles left to themselves, and which, in the case of the vessels, implies the state of dilatation.

¶ 69. *So far from there being anything in the action of the vessels to render it necessary to apply a different interpretation to the action of the heart, the interpretation of the action of the heart which has been given, will, when applied to the action of the vessels, supply an intelligible explanation of "capillary force."*

If arterial blood have the effect of producing dilatation in the vessels by putting an end to that state of contraction which is developed at the time when arterial blood is converted into venous blood, it is plain that arterial blood will favor its own admission into the vessels: if the coats of the vessels be thrown into a state of contraction by the action which is developed in the vaso-motor nerves at the moment when the arterial blood is used up by being converted into venous blood, it is not less plain that there is a cause at work which will help the venous blood out of the vessels. If, indeed, the blood act

in this manner, there may be, it is plain, a state of diastole and systole in the minute vessels which is strictly analogous to the diastole and systole of the heart—there may be, that is to say, a state of things which will readily furnish a physical and intelligible explanation of that independent power in the vessels which evidently co-operates with the heart in carrying on the circulation, and which is generally spoken of under the name of capillary force. At any rate, there is nothing in the action of the vessels which is calculated to cast any doubt as to the soundness of the conclusions which have been drawn respecting the action of the heart.

¶ 70. *The rhythmical movements of the heart appear to be somewhat more intelligible when they are interpreted in accordance with this view of muscular motion.*

It is not for me to say how far these movements become intelligible when they are interpreted by the view of muscular motion set forth in these Lectures, but this I may say—that more is gained than lost by using this mode of interpretation in place of that which is supplied by the current view of muscular motion.

(2) On the peristaltic movements of the alimentary canal.

¶ 71. *The blood would seem to play one and the same part in the movements of the alimentary canal, in the movements of the heart, and in muscular motion generally.*

M. Spiegelberg, of Göttingen,* has performed

* Henle and Pfeuffer's "Zeitschrift," 3 Reihe, ii. 1857.

certain experiments which show that the peristaltic movements of the alimentary canal are increased when the admission of blood into the vessels of the canal is prevented by pressing upon the abdominal aorta, and diminished when the removal of this pressure allows the blood to return to these vessels: and he has also performed other experiments which show that the same movements are increased, though not to the same degree as in the last case, when the intestinal vessels are kept full of venous blood by pressing upon the vena cava or vena porta, and diminished when, by removing this pressure, these vessels are allowed at once to get rid of black blood and to receive red blood. What is, indeed, plainly shown is this—that there is nothing peculiar in the way in which the muscular movements of the alimentary canal are affected by the action of the blood.

¶ 72. *The manner in which the movements of the alimentary canal are affected when the spinal cord or grand sympathetic is subjected to the shocks of a coil-machine need lead to no different conclusion respecting the action of the heart and muscular action generally.*

M. Pflüger* has shown that the peristaltic movements of the alimentary canal of a dog or rabbit are suspended by subjecting the spinal cord or grand sympathetic nerve to electric shocks of a certain strength. Mr. Lister† has repeated these experi-

* "Ueber das Hemmungs-Nervensystem für die peristaltischen Bewegungen der Gedärme." Berlin, 1856.

† "Proc. of Royal Society," 13th August, 1858.

ments, and shown that an opposite result—increase of movement, that is to say, instead of arrest of movement—is obtained by employing weak electric shocks. In all particulars, these experiments agree with those in which the medulla oblongata and pneumogastrics were exposed to the action of strong and weak electric shocks (¶ 66). In these experiments, the peristaltic movements of the alimentary canal are arrested by strong shocks, and accelerated by weak shocks; in those experiments, the rhythmical movements of the heart were arrested by strong shocks, and accelerated by weak shocks. The results are strictly the same, so far as muscular movement is concerned; and, therefore, it may fairly be assumed that the same explanation will serve in both cases.

¶ 73. *The peristaltic movements of the alimentary canal appear to be somewhat more intelligible when interpreted in accordance with this view of muscular motion.*

After what has been said of the action of the nerves and nervous centers of MM. Bidder and Rosenberger in the rhythmical movements of the heart, it may be assumed that the ganglionic structures in the submucous tissue of the alimentary canal, lately detected by M. Meissner, of Bâle,* will discharge a similar office in relation to the peristaltic movements of this viscus. It may be assumed, indeed, that there peristaltic contractions may be traced to certain changing impressions upon the afferent nerves of these gan-

* Henle and Pfeuffer's "Zeitschrift für rationelle Medicin," viii. p. 364. 1857.

glionic structures by substances contained in the alimentary canal, and that these impressions act through the instrumentality of the electricity of the nerves, in the manner already pointed out (¶ 54). And certainly this view is as intelligible as that which supposes that these impressions act through the instrumentality of a vital property of irritability in nerve and muscle.

(3) On the respiratory movements of the chest.

¶ 74. *There would appear to be nothing peculiar in the manner in which the respiratory movements of the chest are affected by "nervous influence."*

The connection of the rhythmical movements of the chest with certain operations in the nervous system, in which the medulla oblongata is the great nerve-center, and the pneumogastric and the phrenic nerves respectively the principal afferent and efferent nerves, is a fact which is not to be questioned. The dependence of these movements upon the respiratory interchanges which take place between the air and the blood in the air-passages, is equally a matter of certainty. Nor need there be much uncertainty with respect to the manner in which these interchanges may produce the nervous action which is necessary to bring about the proper movements in the walls of the chest. For what is the case as interpreted by the view of nervous action set forth in these Lectures? The case in inspiration appears to be this:— that the oxygen of the air makes an "impression" upon the pneumogastric and other afferent nerves by reversing the electrical relations of the exterior

and interior of the nerve-fibers in the part or parts acted upon; that this reversal sets up a state of action in the phrenic and other efferent nerves, of which an electrical discharge, analogous to that of the torpedo, is the accompaniment; that this electrical discharge, analogous to that of the torpedo, produces contraction of the muscular walls of the chest; and that this contraction enlarges the capacity of the chest, and causes a vacuum in the air-passages into which the air rushes. And the case in expiration appears to be this:—that the oxygen of the inspired air disappears in the process of respiration; that this disappearance of oxygen removes the cause which had set up that state of action in the phrenic and other efferent nerves which led to the inspiratory contractions in the muscular walls of the chest; that this cessation of action in these nerves leaves the muscular walls of the chest free to fall into that state of relaxation which is natural to living muscles when left to themselves; and that this relaxation in its muscular walls allows the chest to yield to the pressure of the atmosphere, and to press out, in so doing, the air which had been taken into the air-passages in inspiration. After each expiration, the oxygen of fresh quantities of air will renew the "impression" upon the pneumogastric and other afferent nerves which led to the inspiratory contractions of the muscular walls of the chest; and after each inspiration expiration must follow in due time, as already explained: and in this way, as it would seem, expiration must lead to inspiration, and expiration must succeed to inspiration, as long as the air and the respiratory apparatus are in the conditions necessary to act and react.

¶ 75. *The respiratory movements of the chest do not become more unintelligible when they are interpreted in accordance with this view of muscular motion.*

So far from this being the case, it would seem that a contrary conclusion is inevitable; for a view which refers these movements to the operation of a physical law is more entitled to attention, as supplying a true explanation, than a view which calls in the aid of a vital principle with capabilities of doing anything that may be wanted, and is satisfied with the help of so convenient an agent.

¶ 76. *There is reason to believe that the theory which is applicable to ordinary muscular motion is applicable also to rhythmical muscular motion, and that an insight into the cause of the rhythm is a result of so applying it.*

In the three forms of rhythmical muscular movement which have been considered, there is nothing to contradict and much to confirm the previous conclusions respecting ordinary muscular motion; and the conclusion of the whole matter appears to be that which is stated as the heading to this paragraph. At any rate, I find it difficult, if not impossible, to entertain any other opinion.

¶ 77. *There is reason to believe that the theory of muscular motion set forth in these Lectures derives no small amount of confirmation from the fact that it leads us a step nearer to the discovery of a common law for organic and inorganic nature.*

To my mind, the very strongest argument in favor of the theory of muscular motion set forth in these Lectures is to be found in the fact that it leads us a step nearer to the discovery of a common law for organic and inorganic nature—a law to the existence of which the instincts and the discoveries of science alike bear testimony—a law which does not entomb life in matter, but which quickens matter into life, and surrounds life with a halo of divinity, for it is but a step from the discovery of a common law to the central point in which the immediate operation of the One Divine Lawgiver becomes visible in the law. To my mind, I say, the fact that this theory of muscular motion tends to bind a certain number of vital and physical phenomena together in a common bond is the very *experimentum crucis* in its favor,—and with this remark I bring to close what I have to say upon the physiology of muscular motion.

II. ON THE PHYSIOLOGY OF SENSATION.

In speaking upon the physiology of muscular motion I have excluded all arguments belonging to pure pathology as contradistinguished from pure physiology, and in doing so, I have stated my case in a very lame manner. In speaking upon the physiology of sensation, I am not only at the same disadvantage from pursuing the same course, but I have the additional disadvantages arising from the fact that I have left myself only time for the barest

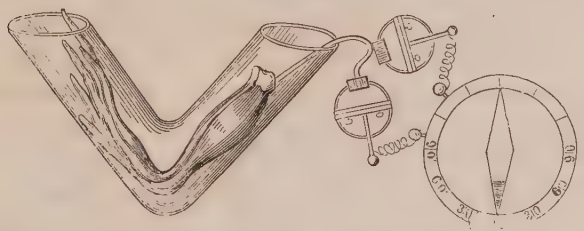
hints of what I intended to say in each division of the plan which I had mapped out for myself.

§ I. On the part which animal electricity has to play in the process of sensation.

¶ 78. *In the case of a sentient nerve, as in the case of a motor nerve, the nerve loses electricity when it passes from the state of rest into that of action.*

The demonstration of this fact is by M. Du Bois-Reymond.* The experiment upon which the demonstration rests is one in which a rheoscopic limb, with the skin not stripped off, is placed, as in the accompanying figure, in a V-shaped tube, with its nerve

Fig. 23.



brought out and laid upon the cushions of a suitable galvanometer. Then, after having waited until the needle of the galvanometer has taken up the position into which it diverges under the action of the current which proceeds into the coil of the instrument from the nerve lying upon the cushions, the V-shaped tube is filled half full of water at a temperature just under the boiling point. This is what is done; what happens is this—that the needle recedes toward zero, as soon as the skin is reached

* "Untersuchungen," vol. ii. p. 520.

by the hot water. The needle, that is to say, recedes toward zero under circumstances which would give rise to keen sensation, if the sentient nerves of the skin of the parboiled limb were in connection with the sensorium; and thus in the case of the sentient nerve, as in the case of the motor nerve, it is plain that the nerve loses electricity when it passes from the state of rest into that of action.

§ II. On the part which artificial electricity has to play in the process of sensation.

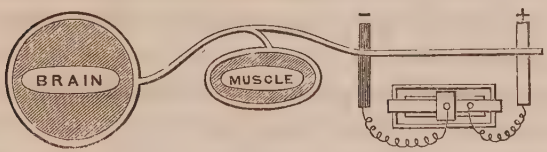
¶ 79. *The change in a sensory nerve when sensation is produced by the action of voltaic electricity, and the change in a motor nerve when muscular contraction is produced by the same means, are exact equivalents.*

M. Matteucci describes* an experiment upon a rabbit which furnishes the proof of this statement. The experiment consists in dividing one of the sciatic nerves low down in the ham, in dissecting out the upper portion of the nerve to a sufficient length, and in placing this upper portion of the nerve across the poles of a voltaic battery. The result is this—that the animal screams with pain and struggles convulsively at the moments of closing and opening the circuit, or at one or other of these moments singly. At first, the screams and the convulsive movements occur equally at the moments of closing and opening the circuit, and it is immaterial, so far as these results are concerned, whether the

* Traité des Phénomènes Electro-Physiologiques," vol. i. Paris, 1844.

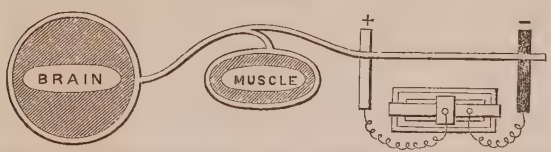
positive pole be next to the brain, or whether the negative pole be in this position. Afterward, the screams and convulsive movements are present at

Fig. 24.



the closing of the circuit, and absent at the opening, when, as in Fig. 24, the negative pole is in the position next the brain; and absent at the closing of the

Fig. 25.



circuit, and present at the opening, when, as in Fig. 25, the positive pole is in the position next the brain. Pain and convulsion, that is to say, come together and go together. This is the plain fact: and this being the fact, there is reason to believe that the presence or absence of pain at the opening and closing of the voltaic circuit will have to be explained in the same way as the presence or absence of the convulsion—a way about which enough has been said in a former lecture (¶¶ 25, 26, 27). In a word, there is reason to believe that the change in a sensory nerve when sensation is produced by the action of voltaic electricity, and the change in a motor nerve when muscular contraction is produced by the same means, are exact equivalents.

§ III. On the conclusions respecting sensation which appear to arise out of the premises.

¶ 80. *There is reason to believe that there is no essential difference between the action which issues in sensation, and the action which issues in muscular contraction.*

Taking the two facts which have been under consideration in the two preceding sections, and interpreting them by what has gone before, there would seem to be good reason for this belief. There would seem to be reason to believe, that is to say, that the action of a sentient nerve may have to be explained, *not* by assuming that a vital property of peculiar irritability has been stimulated into a state of functional activity, but by supposing,—(1) that the "impression" which issues in sensation reverses the electrical relations of the exterior and interior of the nerve-fibers in the part acted upon,—(2) that this reversal leads to a state of action in the sentient nerve,—(3) that this state of action implies the development in and near the nerve of an electrical discharge analogous to that of the torpedo,—and (4) that this electrical discharge gives rise to sensation, if certain ganglionic cells of the sensorium happen to lie near enough to be exposed to its shock. According to this view, indeed, the production of sensation and the production of muscular contraction only differ in this—that the electrical discharge, analogous to that of the torpedo, which is developed in and near the nerve in the state of nervous action, happens to tell upon sensorial ganglionic cells in the one case, and upon muscular fibers in the other case.

¶ 81. *There is reason to conclude that the problems of muscular motion and sensation are only to be solved when the agency of animal electricity is employed as the master-key.*

The whole tenor of the evidence advanced hitherto leads to this conclusion, and to none other. Looking back at this evidence, indeed, we may now see a deep meaning in the words quoted by Humboldt from Brandes, and, without any strain upon the imagination, we may believe that "la physiologie doit à Galvani et à Harvey ses deux bases principales." And looking forward, we may agree with Aldini, Galvani's nephew, in hoping that animal electricity "may one day throw great light on the progress of medicine, and be productive of considerable benefit to the human race."

LECTURE V.

In the four Lectures which yet remain to be delivered, I propose to say something respecting convulsion, tremor, spasm, pain, and paralysis in certain aspects,—the topics, that is to say, to the consideration of which the physiological inquiries of the preceding Lectures have prepared the way; and I hope to be able to show that the lessons in pathology and therapeutics which have yet to be learnt are in strict harmony with the lessons in physiology which have been learnt already.

I shall form my conclusions respecting the pathology of convulsion, tremor, spasm, and pain, from the condition of the functions of respiration, circulation, and innervation in each of these disorders. My principal object will be to ascertain whether the disorder under consideration is associated with exalted vitality or with depressed vitality; and I think I shall attain this object by following this plan; for it must be evident that the activity or inactivity of these three great functions must furnish a correct measure of the degree of vitality in the system at the time.

In the remarks I have to make upon the therapeutics of the four disorders which have just been named, and upon paralysis in certain aspects, I must be very brief. Indeed the time remaining at my dis-

posal at the conclusion of the pathological portion of the inquiry upon which I am now entering will not permit me to do more than to offer a few general and hasty observations upon these topics.

III. ON CONVULSION.

§ I. On the pathology of convulsion.

I CANNOT stay to describe the various forms in which convulsion may be manifested. I cannot stay to describe simple epilepsy, or the epileptiform disorders which may be connected with different diseases of the brain—chronic softening, chronic meningitis, tumor, induration, atrophy, congestion, apoplexy, inflammation, and the rest—with fever, with certain suppressed excretions, with "irritation" in various organs, and with the dying state. I cannot stay to describe convulsion as it occurs in hysteria, in chorea, or in those strange affections which take an intermediate position between the two, as in the dance of St. Vitus and St. John, in tarantism, and in other affections of the kind. All I can do, indeed, is to take a cursory general survey of the history of convulsion as it is commonly seen in epilepsy, in epileptiform disorder, in hysteria, and in chorea, with a view to catch the common and constant features of the disorder.

(1) On the pathology of convulsion as deduced from the condition of the respiration in this disorder.

¶ 82. *The epileptic and epileptiform paroxysm is not unfrequently preceded by signs of defective respiration.*

The evidence upon this point is, perhaps, not very conclusive. I think, however, that the habit of sighing, which is not very unfrequent among epileptics, and which is often most perceptible when a fit is at no great distance, may be looked upon as a sign that a certain amount of want of breath has to be made up now and then by some breaths which are more deeply drawn than usual. I have also notes of more than one case in which, when the fits happened during sleep, the movements of the chest often came to a complete stand-still for a few moments before the convulsions began—a stand-still so complete as to have led the looker-on to fear that death had actually gained the victory.

¶ 83. *The epileptic or epileptiform paroxysm is usually accompanied by a state of unmistakable suffocation.*

The livid, black, and bloated head and neck, the sounds suggestive of strangling, the evident suspension of all proper respiratory movements, are symptoms which explain themselves; and these are the symptoms which are usually present in the fully-developed form of the epileptic or epileptiform paroxysm. They suggest the idea of death by the bowstring of some invisible executioner. Nor is the case really different in those varieties of general or partial epileptic or epileptiform disorder in which the face remains pale and shrunken from the beginning to the end of the paroxysm: for in these cases there is a ghastly lividity of the countenance which shows very plainly that the convulsive symptoms are accompanied by some grave interruption in the process of respiration.

¶ 84. *The convulsion of hysteria or chorea is associated with a state of very defective respiration.*

In these forms of convulsive disorder the breathing is not arrested as it is in the epileptic or epileptiform paroxysms, but it is shallow, embarrassed, often prolonged into sighs, and generally accompanied by a distressing sense of want of breath. It is hampered and interrupted in no inconsiderable degree.

¶ 85. *The condition of the respiration during convulsion is one which supports the notion that the convulsion is connected with depressed and not with exalted vitality.*

This is a necessary conclusion to what has just been said, if, as must needs be, the amount of vital activity is to be measured by the amount of respiratory activity.

(2) On the pathology of convulsion as deduced from the condition of the circulation in this disorder.

¶ 86. *In the chronic forms of convulsive disorder the inter-paroxysmal condition is usually marked by evident signs of a feeble circulation.*

The inter-paroxysmal state in many cases of *common epilepsy* is marked by a weak and slow pulse, by cool, cold, pale, bluish or congested hands and feet, and by an almost habitual feeling of chilliness. Indeed, so far as my own experience goes, the circulation in this state is always wanting in true power, not only in common epilepsy, but also in all the *chronic* forms of epileptiform disorder. In certain *acute* forms of epileptiform disorder, it is true, the

inter-paroxysmal state may be marked by excitement of the circulation; but this fact, as will be seen presently, is in no way calculated to set aside the conclusion which seems to be necessary with respect to the state of the circulation between the fits of common epilepsy, and between the fits of chronic epileptiform disorder.

The pulse of persons who suffer from hysterical convulsion is generally soft, quick, and unequal. The heart is readily thrown into a state of annoying and distressing palpitation, especially by any agitation of the feelings. The hands and feet are scarcely ever warm, and there is a disposition to chilblains, even when the weather is not very cold. Everything, indeed, goes to show that the circulation is wanting in healthy vigor.

Nor is it otherwise in chorea. A disposition to rheumatic fever would seem to be not uncommon in this affection, at least in this country; but it is not to be supposed on this account that the febrile and the choreic symptoms are in any way concurrent. On the contrary, there is usually a wide interval of time between the latter symptoms and the former; and thus the predisposition to rheumatic fever, if it exist, cannot be taken as an objection to the fact, now very generally admitted, that chorea is essentially a feverless malady. Indeed, this very predisposition may be taken as an argument that the circulation in chorea is below the normal standard of activity: for is it not a fact that the heart is often damaged by valvular disease, that the "capillary power" is often very defective, and that the circulation, from one cause or another, is always more or

less feeble in persons who are liable to attacks of rheumatic fever?

In the chronic forms of convulsive disorder—common epilepsy, chronic epileptiform disorder, hysteria and chorea,—there would seem, indeed, to be good reason to conclude that the inter-paroxysmal condition is marked by evident signs of a feeble circulation; and, therefore, I am not hasty in accepting this conclusion as that which ought to be adopted.

¶ 87. *The epileptic and epileptiform paroxysm is usually, if not invariably, ushered in by signs of failure in the circulation.*

The immediate precursor of the perfect form of the paroxysm is a sign which is somewhat difficult to catch—corpse-like paleness of the countenance. M. Delasiauve* was the first to notice this phenomenon: and M. Trousseau insists upon it as a mark which distinguishes true epilepsy from feigned epilepsy. "Il est une signe," he says, "qui se produit du moment de la chute, et qui n'est imitable pour personne; c'est la pâleur très-prononcée cadavérique, qui couvre pour un instant la face épileptique. Nous ne le voyons pas, parceque nous arrivons toujours trop tard, alors que la face est déjà d'une rouge très-prononcée."† In a word, the general form of the epileptic or epileptiform paroxysm begins in the same way as the partial form: for it is allowed by all that the initial symptom in the latter case is cadaverous pallor of the countenance. I have often seen this peculiar symptom: and, in addition, I have

* "Traité de l'Epilepsie," 8vo. Paris, 1855.

† "L'Union Médicale," 28 Avril, 1855.

always found it associated with great feebleness of the pulse at the wrist and elsewhere.

¶ 88. *At the height of the epileptic or epileptiform paroxysm the pulse is usually full, strong, and frequent, because the arteries are then laboring under a load of* BLACK *blood, as they are found to labor in suffocation, and not because these vessels are then receiving an increased supply of* RED *blood.*

In some cases the pulse at the wrist is almost or altogether imperceptible from the beginning to the end of the paroxysm: in other cases it rallies speedily, and, when the fit is at its height, it beats with considerable force, fullness, and frequency. How, then, is this? What is the true meaning of this vascular reaction? The current belief on the subject is that an increased quantity of *red blood* is injected into the arteries during the convulsion, and that this increased quantity of red blood produces the convulsion by provoking a state of increased vital activity in one or other of the great nervous centers; and very recently the late Professor Schroeder van der Kolk has given distinct expression to this belief.* In reality, however, there is reason to believe that the pulse acquires power in the epileptic or epileptiform paroxysm because the condition of the circulation at the time is one of suffocation, and for this reason simply. For, what is the condition of the circulation in suffocation? It is *not* one in which, as is generally supposed, the arterial pulse rapidly fails for want of blood, and the venous sys-

* "On the Proximate Cause and Rational Treatment of Epilepsy." New Sydenham Society Series, 8vo. London, 1859.

tem as rapidly becomes gorged with unaerated blood; on the contrary, it is one in which the arterial system becomes gorged at the expense of the venous system, and in which the pulse in the arteries becomes stronger and stronger as the blood within these vessels becomes more and more venous in its character. All this is proved experimentally by the late Dr. John Reid,* of Aberdeen, and by Professor Draper,† the younger, of New York. In a rabbit in which he had laid bare the great vessels of the neck and tied the windpipe, Dr. Reid saw the blood in the carotid change from red to black as the process of suffocation went on, until its color became undistinguishable from that of the black blood in the neighboring jugular. He saw this change of color taking place in the stream of blood within the unopened vessel, for the coats of the carotids and jugulars are passably transparent; and he saw it more plainly still in a jet of blood from an artificial opening in the artery. He also measured the force of the pulse in the carotid by the hæmadynamometer, and found that it became greatly increased, perhaps doubled, during the time that the process of suffocation was in progress. In other words, he discovered that black blood finds its way into the arteries during the process of suffocation, until it has displaced all the red blood, and that, for awhile, the force of the pulse augments progressively and considerably under these circumstances. And in a rabbit in which he had opened the chest and then closed

* "Phys. Anat. and Pathol. Researches," 8vo. Edinburgh, 1848.

† "Lectures on the Physiology of the Circulation," in Amer. Med. Monthly, April, 1860.

the windpipe by a ligature, Dr. Draper saw the blood leaving the right side of the heart and the vena cava, and accumulating in the aorta and in the left side of the heart (in the aorta first in order) as the process of suffocation made progress: and he also found that this state of things did not pass off immediately after the death of the animal—that it continued, in fact, until the arterial side of the heart and the arteries had had time to empty themselves in the ordinary way. Here, then, are facts (for facts they assuredly are) which show that the powerful pulse which may be present during the epileptic and epileptiform paroxysm may have a very different significance to that which is usually attached to it. Here, indeed, are facts which show that this powerful pulse may be nothing more than the natural pulse of the process of suffocation, the pulse of black blood—the *apnœal pulse*, as it may be called; for it has been seen that the convulsion in which the pulse is full and strong and frequent is accompanied by a state of unmistakable suffocation. Nay, this is the only conclusion which can be drawn; for with the respiration completely arrested, as it is, it is simply impossible that there can be an increased injection of *red* blood into the arteries during the paroxysm. Nor is it only with reference to the condition of the pulse in convulsion that these facts are of interest. On the contrary, they explain many apparent anomalies in the circulation. For example, they explain how it is that the blood drawn from the temporal artery in a fit has often been black in color, and projected to an unusual distance; and how, in cases of congestion of the lungs, and in some other

cases where the areation of the blood is greatly interfered with, the pulse may beat with seemingly contradictory power in the very last moments of life. They show, in fact, that the pulse may derive a fictitious power from admission of black blood into the arteries, and that mere power of pulse, apart from the condition of the respiration, may be a very unsafe criterion of vital power.

¶ 89. *Convulsion is never coincident with a state of active febrile excitement of the circulation.*

In the fevers of infancy and early childhood, especially in the exanthematous forms of these disorders, convulsion not unfrequently occupies the place which belongs to rigor in the fevers of youth and riper years. It occurs in the cold stage of the fever, when the powers of the circulation are greatly depressed in every way; and it is confined to this stage, except there are certain brain or kidney complications, of which more will have to be said presently. In a word, it never occurs during the hot stage of the fever, when the circulation is carried on with undue vigor, or during the stage of collapse, when the powers of the nervous system are altogether exhausted.

I think it will also be found that convulsion holds the same relation to the sympathetic fever which is associated with inflammation of the brain or its membranes, or with inflammation in any other part; for the constant rule in these cases appears to be, that the convulsion is connected with the cold stage before the hot stage, or with the cold stage after the hot stage, and never with the hot stage

itself. Nay, I am even disposed to think that there is something altogether uncongenial between convulsion and the hot stage of the sympathetic fever connected with inflammation: for it is a fact, not unfrequently verified, that the fits of common epilepsy are often suspended for the time by causes which give rise to a state of sympathetic fever in the system. For example, I can call to mind four cases of epilepsy in which a great deal of sympathetic fever was produced by a burn or fracture inflicted during a fit, and in which fits, which were of daily occurrence before the accident, and which recurred with the same degree of frequency afterward, were altogether suspended so long as the fever continued.

And certainly there is nothing to lead to a contrary conclusion in the history of the convulsion which may be connected with teething, with worms, or with some other condition in which what is called "morbid irritability" is the prominent characteristic; for it is found on inquiry, not only that fever is almost entirely foreign to the state of "morbid irritability," but also that convulsion, when it does occur, is associated with the periods of considerable vascular depression, and not with the periods of inconsiderable vascular reaction.

In a word, the result of bedside study has convinced me that the true place of convulsion in connection with any form of febrile disorder is in the cold stage before the hot stage, or in the cold stage after the hot stage, and never in the hot stage itself —that, in fact, there is something uncongenial between convulsion and an excited condition of the circulation.

¶ 90. *The convulsion which may attend upon the close of Bright's disease is connected with a pale and watery condition of the blood, and with unmistakable signs of great vascular debility, as well as with suspicions of uræmic poisoning.*

It is not easy to theorize upon the way in which convulsion is brought about when urea is retained in the blood, or rather when this retained urea is resolved into carbonate of ammonia in the blood. The simple fact appears to be, that the powers of the circulation become more and more enfeebled as the blood becomes more and more contaminated, and that they are reduced almost to the last degree of feebleness when the convulsion happens; but how to explain the fact is by no means evident. It may be that the uræmic poisoning acts by destroying the blood-corpuscles. Or it may be that the great deficiency of blood-corpuscles, which is a marked characteristic of Bright's disease in its advanced stage, is independent of uræmic poisoning, and more concerned in the production of the head-symptoms than the uræmic poisoning. Dr. Watson is of opinion that the pale and watery condition to which the blood is at last reduced in albuminuria may have something to do in bringing about the stupor and coma of the ending scenes of the disorder; and he bases this opinion upon the fact that similar symptoms are apt to ensue, in conjunction with a similar deficiency of hæmatosin, in spurious hydrocephalus: and I am quite disposed to subscribe to this opinion, and to apply it to the interpretation of the convulsion as well as to the interpretation of the stupor and coma. But upon these

points, and upon all others connected with them, I must refrain from dilating at present.

¶ 91. *Epileptiform convulsion is a direct consequence of sudden and copious loss of blood.*

This fact has already been sufficiently commented upon wher speaking on the part which the blood has to play in the physiology of muscular motion (¶ 35). It can, indeed, have only one significance.

¶ 92. *The condition of the circulation during convulsion is one which supports the notion that the convulsion is connected with depressed, and not with exalted, vital power.*

All this follows necessarily from what has gone before, if it only be allowed, as it must needs be, that the degree of vitality is in direct relation to the activity of the circulation.

(3) On the pathology of convulsion as deduced from the condition of the innervation in this disorder.

¶ 93. *The signs of wanting brain-power are scarcely ever absent in persons who are liable to epileptic and other chronic forms of convulsive disorder.*

In very many cases of epilepsy there is a want of fire in the countenance, and a dilated and sluggish state of the pupil, which seem to point to the brain as lacking in energy; and, in keeping with these signs, it is found on inquiry that the memory is more or less treacherous, the understanding more or less vacant and listless, the feelings more or less insubordinate. It is, no doubt, easy enough to meet with epileptics who, without any want of candor on

their part, will deny the existence of any flaw in their mental faculties, and who have all but an absolute right to do so; but in cases of ordinary severity I do not remember a single instance in which this denial was fully borne out by the testimony of the friends of the patient. And in a case of this kind the testimony of friends is more to be relied on than the testimony of any patient.

In many confirmed and aggravated cases of epilepsy a short examination will show that a terrible blight has fallen upon all the faculties which distinguish man from the mere animal. In these cases, indeed, a single glance at the countenance will often serve at once to detect this blight, and to connect it with epilepsy; for the features tell a story of dullness and gloom which is almost characteristic of the disease, even though the skin of the eyelids and temples presents none of those crimson specks which are certain signs that the face has been "black and full of blood" in some recent paroxysm. In the health of the mere body there may be but little wrong—nothing beyond a somewhat feeble circulation and a somewhat insufficient respiration: in the health of the mind the case is altogether different; and, therefore, it is to be supposed that the obscure traces of mental imperfection which are present in cases in which the characteristics of the disease are not fully marked, are in reality not accidental, but essential, and that no account of the inter-paroxysmal state in these cases would be complete which did not include them.

In all cases, as might be expected, the evidence of mental imperfection is most apparent after a fit.

At this time, indeed, the faculties of the mind may be so blunted that the features of the epileptic may become blended with those of the demented person; or symptoms of intellectual or moral aberration may show themselves, and the epileptic for the time may be transformed into the lunatic. The fits, also, may recur so frequently that the mind may never have the chance of clearing up in the interval, and in this way the distinctive characters of the convulsive malady may become confounded with those of dementia and insanity. Not unfrequently, also, there is the very gravest degree of mental infirmity from the very first, and instead of only tending to dementia, the history of the epileptic may begin in sheer idiotcy. Indeed, it cannot be looked upon as a mere accident, that idiotcy and epilepsy should so often go together, and that the head of the epileptic should be so frequently wanting in proper size and proportions as to suggest to the least imaginative observer its suspicious kinship to the head of the idiot.

Nor is it an objection to this view that men like Julius Cæsar or Napoleon I. should have had epileptic seizures; for who can say that the brains of these men were in a perfectly healthy condition when these seizures made their appearance?

And certainly there is no lack of evidence to show that the subjects of hysterical or choreic convulsion are in all respects the very reverse of "strong-minded."

They who suffer from what is ordinarily called hysterical convulsion belong almost exclusively to the female sex; and they present in addition, and in an aggravated degree, the peculiar weakness of

this sex. For the most part, they are undecided, irresolute, fickle, purposeless, yielding easily and almost passively to every impulse, either from within or from without, and scarcely ever capable of anything like sustained action. They do what they ought not to do, and they leave undone what they ought to do, and their excuse is that they cannot help it. With them *will* is little more than an empty name, and so much are they the creatures of feeling, that a small matter serves to make them melt into tears, or burst into laughter. The temper, also, is as little under control as the feelings, and impatience, perverseness, obstinacy, and anger are no uncommon symptoms. There is no lack of imagination; but, as a rule, the ideas are allowed to take their own course, with little check from the reason; and hence, fancies and whims of all kinds in endless succession, or, what is worse, some one whim or fancy in possession of the mind, and the reason unable to eject it. Not unfrequently, also, there is a disposition to exaggeration and deceit, which must betoken some bluntness in the moral sense, if some allowance have not to be made on the ground of an imagination which cannot always stoop low enough to perceive the line which separates facts from fancies. These mental peculiarities are all written with a certain degree of plainness upon the countenance. There is no want of brightness in the eye, no sluggishness in the pupil, no marks of "slowness," as in many epileptics. On the contrary, there is a brisk, unsteady expression, which shows that the mental error is on the side, not of dulness, but of sensitiveness.

The persons who suffer from chorea agree in mental peculiarities with those who suffer from hysterical convulsion. They exhibit, often in an exaggerated degree, the same signs of wanting will, of halting reason, of inordinate sensitiveness. They suffer from the same timidity, the same fretfulness, the same uncertainty and irritability of disposition and temper. If there be any marked difference, it is that they have less vivacity. In many cases this want of vivacity is written upon the countenance in an expression of languor and vacancy; in some cases this expression may be so marked, that a person suffering from chorea, if seen in a moment of quiet, may be mistaken for an idiot. Indeed, it may be necessary, in these latter cases, to make the patient get up and move about before the true nature of his malady becomes apparent.

It is impossible to enter into further particulars without sacrificing time which cannot be spared. Much more might have been said, but I trust enough has been said to show that signs of a weak or imperfect or jaded brain are scarcely ever absent in persons who are liable to epileptic and other chronic forms of convulsive disorders, and therefore I pass on to the next topic without further delay.

¶ 94. *All signs of mental life are abolished, or all but abolished, during the paroxysm of convulsion.*

In general convulsion of an epileptic or epileptiform character the mind is a perfect blank, and so also, with very few exceptions, in the partial forms of the same disorders. In the convulsion of hysteria, and in the more severe forms of choreic disturbance,

the will is altogether in abeyance, and the intellectual state is one which approaches very closely to that of unconsciousness. Hence it is not to be supposed that convulsion is in any way connected with exalted functional activity of that part of the brain which ministers to the mental faculties.

¶ 95. *There is no clinical evidence to show that convulsion is associated with an overactive condition of the circulation in the brain.*

There is little reason to believe that convulsion is ever present in the hot stage of active inflammation of the brain or its membranes—a state in which there is general feverishness, and what is called active "determination of blood to the head." There is, in fact, much evidence to the contrary. When convulsion attends upon these disorders, it either takes the place of rigor in the cold stage before the hot stage, or else (much more rarely) it replaces subsultus in the cold stage after the hot stage, when the patient has all but ceased to strive in the "struggle called living."

There are, however, certain cases in which convulsive movements are associated with a state of unequivocal determination of arterial blood to the head, and with considerable reaction of the circulation generally. Yesterday, for example, I saw a case in point. The patient was a lady, upwards of sixty years of age, who for two years previously had been demented, epileptic, and partially hemiplegic. Ordinarily her circulation was miserably feeble, and her hands and feet cold and comparatively bloodless. Early yesterday morning, about seven o'clock or so,

she left her bed with a view to relieve her bowels; and after straining ineffectively for some time, she made signs which gave her maid to understand that she had pain in her head, and went to bed again. Shortly afterward, the maid found her unconscious, and in one of her old fits—"rather a sharp one," the comment was. I was called in about four hours after this time, and I was much interested in what I saw. The patient was altogether unconscious, and every few moments she was shaken with convulsive movements, and this had been her state ever since the first fit. The convulsive movements were confined to the features, to the right leg, and to the left arm (it was the right half of the body which had been partially hemiplegic), the rest of the body—the right arm, the left leg, the muscles of the abdomen, and the muscles of the chest especially, being almost or altogether unaffected. There was a good deal of phlegm in the mouth and throat, and much choking noise arising therefrom, but the lips and complexion were red and without any tinge of blueness whatever. Moreover,—and this is the point of present interest,—the skin generally, and the skin of the head especially, was hot and flushed, and the pulse everywhere, but in the carotids most of all, full and throbbing. The pupil, also, was contracted in both eyes. I stood by the bedside for some time. The convulsive movements recurred every few moments; they were not very severe; and they were, without doubt, accompanied by a state of unequivocal determination of arterial blood to the head, and of vascular reaction generally—a state contrasting in all respects with the

ordinary condition of the circulation in the patient. How, then, is this state of things to be accounted for? The symptoms, it is plain, indicated apoplexy into a softened portion of brain in or near the motor tract of nerve-fibers—a state implying laceration of these fibers, and probable pressure upon them. Hence, there is no great obscurity as to the starting-point of the convulsion, for it has been seen that mechanical injuries of various sorts have the effect of producing that reversal in the electrical relations of the exterior and interior of the nerve-fibers, which, according to the physiological premises, is the essential cause of the state of "irritation" (¶ 50). And from this starting-point it is not difficult to go a step further, and see why, sooner or later, a state of vascular reaction, such as is manifested after a fit of epilepsy, or, more plainly still, in the hot stage after the cold stage of ague, *must* follow the state of "irritation" invaso-motor nerves if only this state be carried on long enough to exhaust or paralyze these nerves, and so leave the vessels free to relax and receive more blood. It is not difficult to see that "irritation" in the vaso-motor nerves (of which contraction in the vessels is the sign) may end by leaving the vessels in a state of relaxation, which may soon become a state of reaction; but it is very difficult to believe that this state of vascular reaction is the cause of convulsion. On the contrary, the facts would seem to show that this state of vascular reaction has more to do in antagonizing convulsion than in producing it. It is certain that the convulsive movements which I witnessed in the case under consideration were partial and not very

severe: it is more than probable that the fit which ushered in those movements, which is described as "rather a sharp one," and which, no doubt, preceded the establishment of the state of vascular reaction in the head and elsewhere, was both general and violent; and, therefore, it may be supposed that in this case the convulsion had been antagonized rather than favored by the state of vascular reaction. It is certain that epilepsy may occur in its most violent form in a state of circulation which is closely akin to that of syncope; it is also certain that fits of epilepsy are apt to recur if the circulation does not rally with sufficient promptness after a fit: and, therefore, it may be supposed that convulsion is antagonized rather than favored by the state of vascular reaction. As it seems to me, indeed, there is no reason to believe that an overactive condition of the circulation in the head or elsewhere is a cause of convulsion and much to the contrary; and the only conclusion which I can draw from the case under consideration is—that that particular condition of the nerves which is called "irritation," and of which convulsion is one of the signs, can coexist with a much more active condition of the circulation than that which is usually associated with it,

In order to be fully understood, a case like this must not be taken alone. It must be put in its true place in the history of convulsion, and interpreted accordingly. And if this be done, I think it must be conceded that it will afford no ground for the notion that an overactive condition of the circulation in the brain is associated with convulsion. And this case, so far as my own experience goes, is

the only case which can give any real countenance to such a notion.

¶ 96. *The appearances after death are not calculated to show that convulsion is necessarily connected with inflammation of any one of the great nerve-centers.*

The morbid appearances after death from ordinary epilepsy are very obscure, if the case have really not been one of epileptiform convulsion connected with some special disease. In cases fatal during the fits, the brain has been found to be congested; but this appearance is clearly owing to the mode of death, and it is allowed to be so. In cases where epilepsy has been complicated with insanity, the brain or its membranes may present various signs of inflammation, or of changes more or less akin to inflammation; but these signs are clearly referable to insanity rather than to epilepsy, and for this reason,—that they are as common, or more common, in insanity without epilepsy. In other cases there are signs of degeneracy of the brain, such as pallor of its gray matter, softening, granular induration, atrophy, dropsical effusion—the very signs, indeed, which belong to the demented state. And it is this fact which furnishes some ground for supposing that signs of this character, and not signs of inflammation, may have something to do with epilepsy. For is it not true that the demented state is intimately connected with the epileptic disorder? And is it not equally true that a demented person is almost sure to be affected with palsied shakings, or cramps, or spasms, in one form or another, if he

escape the graver affliction of epilepsy? In other cases, again, the skull may be thicker and heavier than usual, and the several internal projections, the clinoid processes for example, may be considerably developed, or various parts of the dura mater may be ossified; but there are in the brain proper or its membranes no changes of sufficient constancy to be necessarily connected with epilepsy—not even that change in the pituitary body of which so much was said by Wenzel;* for, writing of it, Professor Rokitansky† says that he has "frequently failed to discover it in those who had notoriously suffered from epilepsy and convulsions," and that he has "met with it in those who were perfectly healthy." It is in the medulla oblongata alone, indeed, that there appear to be any changes after death which have any pretensions to constancy. In early cases of epilepsy, it is true, this organ may present no signs of disease: in confirmed cases, on the other hand, it is often hardened by the interstitial deposit of a minutely granular albuminous matter, or else softened, swollen, and presenting signs of evident fatty degeneration. The posterior half of the organ, moreover, is redder and more vascular than it ought to be, even when the patient has not died in a fit; and, on making a more minute examination of this part, the blood-vessels are seen to be dilated to twice their natural dimensions, and with their walls much thickened—in the course of the hypoglossus nerve

* "Beobacht, über den Hernauhang fallsüchtiger Personen," &c. 8vo. Mainz, 1810.

† "Manual of Pathological Anatomy." Translated for the Sydenham Society by Dr. C. H. Moore. Vol. iii. p. 434.

and corpus olivare, in epileptics who were in the habit of biting their tongue in a fit, and in the course of the roots of the vagus in epileptics who were not in this habit.* These facts, for the knowledge of which we are indebted to the late Schrœder Van der Kolk, are not altogether intelligible, and they must show, if they show anything, that the medulla oblongata of the epileptic is damaged in structure, and in a proportionate degree rendered incapable of discharging its proper offices efficiently. The signs of fatty degeneration have but one significance. The interstitial deposit, also, implies an equivalent absence of healthy nerve-structure, and so, in a measure, does the dilated condition of the blood-vessels. In a word, the appearances after death in the medulla oblongata of epileptics are more in accordance with the notion that epilepsy is connected with a depressed state of functional activity in this organ, than with the contrary notion.

Nor are the disclosures of pathological anatomy in chorea such as to connect this malady with an inflammatory condition in some one or other of the great nerve-centers. In fourteen cases of fatal chorea contained in the late Dr. Hughes' excellent memoir† on this disease (which cases may be said to constitute more unexceptionable evidence on the subject than any others on record), the brain was quite healthy in four, and only congested in three others; so that it is necessary to conclude that there was nothing

* "On the Proximate Cause and Rational Treatment of Epilepsy." Translated for the New Sydenham Society, by Dr. C. H. Moore. 1859.

† "Guy's Hospital Reports," 1846. Second series. Vol. iv. p. 372.

the matter with the brain in half the cases. And of the remaining seven cases, the particulars are as follows: in the first, serous effusion beneath the arachnoid, and into the ventricles, slight effusion of blood beneath the right cerebral hemisphere, softened brain; in the second, arachnoid opaque, brain dark and soft; in the third, pia mater watery, cineritious matter red, soft, and partially adherent; in the fourth, brain soft and vascular, much fluid in ventricles; in the fifth, arachnoid opaque in parts, cerebrum vascular, left thalamus rather soft; in the sixth, dura mater adherent very firmly to calvarium, and more opaque than natural, cerebral vessels turgid; in the seventh, blood effused into arachnoid, fornix, and edge of third ventricle soft, red and tumid, brain softened. In the same fourteen cases the spinal column was not opened in six. In the remaining eight cases the cord and its membranes were quite healthy in three, and only a little congested in one; so that there was nothing the matter with the spinal cord or its membranes in half the number examined. And of the remaining four cases, the particulars are these: in the first, soft adhesions of the arachnoid, gray matter dark; in the second, vessels rather large and numerous, serous surfaces opaque, old adhesions of the membranes, especially behind the cord; in the third, medulla slightly softened, rachidian fluid opaque, yellow and densely coagulable by heat; in the fourth, softening of the cord opposite the fourth and fifth dorsal vertebræ. In half the number of cases, therefore, there are signs which show more or less clearly the presence of inflammatory changes, and in the remaining half there

are no such signs. What then? What is the true relation of these traces of inflammation to the chorea? This is the question, and this is a question which must be left on one side until after the relation of the state of irritation to that of inflammation has been inquired into. In the mean time, however, it is plain that the absence of traces of inflammation in half the cases examined is a plain proof that chorea, like epilepsy, is not necessarily connected with inflammation in any one of the great nerve-centers. And if this be so with respect to chorea and epilepsy, there is no reason to suppose that a contrary conclusion is necessary with respect to hysteria and other forms of convulsive maladies.

¶ 97. *Convulsion must not be looked upon as a symptom of a congested condition of the cerebral veins.*

As it seems to me, the clinical history of disease is opposed to the theories which ascribe convulsion to a congested condition of the cerebral veins. In whooping-cough these veins are often congested in a very high degree during the paroxysm, and yet convulsion is only an accidental accompaniment of the paroxysm. In congestion of the lungs, also, these veins are greatly gorged with black blood; and the consequences of this engorgement are dreamy sleepiness, stupor, perhaps coma, rarely convulsion. Nor is the case different where extreme venous congestion of the brain is brought about by straining or in any other way; for here the symptoms are coma and paralysis, not coma and convulsion; apoplexy, not epilepsy. Moreover, the recent experiments of MM. Kussmaul and Tenner* show very plainly that

* Op. cit.

the effect of tying the internal and external jugulars of rabbits is *not* to convulse these animals, but only to stupefy them for the first twenty-four or thirty hours, and, in some instances, to cause them to gnash their teeth for a short time. Indeed, there is nothing in all this evidence, physiological or clinical, to nullify the conclusion—that venous blood has no special action in producing convulsion.

¶ 98. *The peculiar condition of the nervous system which is known under the name of "irritation," and which in a majority of cases has a great deal to do with the production of convulsion, is in no sense the equivalent of inflammation.*

When speaking upon the physiological part which the nerves have to play in the process of muscular motion, I came to the conclusion that the state of nerve which is called *irritation*, may be the result of a partial reversal in the electrical relations of the exterior and interior of the nerve-fibers at the point from which the irritation sets out. I showed that this partial reversal would put an end to the state of static electrical tension which is the state of nerve during rest, and bring about in its stead a state of electric discharge in and near the nerve—that, in fact, it would have the effect of causing the nerve to pass from the state of rest into that of action (¶ 47). I showed, also, that the partial reversal in question may result from deficient supply of arterial blood, and from mechanical injuries of various sorts (¶¶ 49, 50, 51).

If, then, a nerve in a state of irritation is a nerve in a state of action, and if the state of action may

be accounted for in this manner, it is not difficult to see how some of the results of irritation may find their explanation. It is not difficult to see how the state of irritation may issue in tremor, convulsion, or spasm, in morbid sensations of various kinds, and in a contracted condition of the vessels, according as it may happen to affect the parts of the nervous system which minister to ordinary muscular movements, to common or special sensations, or to the movements of the vessels.

Nor is it difficult to go a step further and see how a continuance of the same irritation may issue in very opposite results, namely, in paralysis of ordinary muscles, in anæsthesia or some analogous condition of special sensation, or in congestive and inflammatory changes. It is not difficult to see how paralysis of the ordinary muscles, or anæsthesia or some analogous condition of special sensation, may result from a continuance of the irritation, for upon any theory of nervous action it is to be supposed that the continuance of the state of action, which is the state of irritation, will eventually deprive the nerve of its irritability. And so likewise it is not difficult to understand how the continuance of the state of irritation in the vaso-motor nerves will issue in congestion or inflammation, for when this irritation has been carried to a point which deprives the nerves of the irirritability, the state of action which previously kept the vessels in a state of contraction is at an end, and the vessels thus left to themselves will relax and receive more blood. The case, indeed, is precisely the same as that which is exhibited in the experiments upon the vaso-motor nerves of the neck (¶ 68),

with this difference only, that in the experiments the state of paralysis which brings about the state of vascular engorgement is produced by *dividing* the vaso-motor nerves, whereas in the case under consideration, the paralysis leading to the vascular engorgement is brought about by keeping the nerves in a state of action or irritation until they have lost their irritability.

According to this view, then, the state of irritation is in no case to be confounded with the state of inflammation. According to this view, the state of irritation would seem to involve a contracted state of the vessels—a state which is even antagonistic to inflammation. It would seem as if the state of vascular contraction arising from irritation were the precursor of the state of inflammation. It would seem, in fact, as if the state of inflammation must follow the state of vascular contraction arising from the irritation, if the irritation have been kept up long enough to deprive the vaso-motor nerves of their irritability, and, in this way, to leave the muscular coats of the vessels free to fall into that state of relaxation which is natural to them when they are left to themselves.

And if this view of the relation of irritation to inflammation be the true view, it is easy to understand how, in some cases of chorea, there may be traces of inflammatory action in some of the great nerve-centers, and not in other cases; for in the cases where these traces are present, all that is necessary is to suppose that the irritation in the vaso-motor nerves has gone on long enough to paralyze the nerves, and leave the vessels free to relax and receive

more blood, and that the irritation has not advanced to this point in the cases where the traces of inflammation are absent. Moreover, it is easy to believe that the traces of inflammation, when present, may be not only in various parts of the nervous system, but in any part of the body, for the arrangement of the nervous system is such as to allow the irritation to be transmitted anywhere, everywhere. Nay, it may even be imagined that the stage of inflammation is one which must put an end to the precursory stage of irritation, for it has been seen (¶ 95) that convulsion is antagonized rather than favored by the state of vascular reaction.

According to this view, then, "irritation" is in no sense the equivalent of inflammation. According to this view, indeed, the *hot stage* of inflammation, with its congested and inflamed vessels, *may follow the cold stage*, with its rigors and pains, and with its shrunken and contracted vessels, because the hot stage is not produced until the "irritation" which produces at one and the same time, and by one and the same means, the rigors and pains and empty vessels of the cold stage, has by its continuance at last brought about a certain amount of paralysis in the nerves belonging specially to the vessels. And certainly there is nothing in the history of convulsion which is contradictory to this view of the relations between "irritation" and inflammation. For is it not true that epilepsy may be present in its most violent forms where it is impossible to associate the "irritation" upon which the convulsion depends with the faintest blush of inflammation in any part of the nervous system or elsewhere? And has it not just

been seen that the epileptiform convulsion which may be associated with cerebral inflammation is anterior or posterior to this inflammation—is, in fact, a substitute for rigor or subsultus?

¶ 99. *The phenomena of aura or globus are not at variance with the premises.*

The phenomenon of globus is more readily explained by referring it to depressed innervation than to a contrary state of things. And certainly it is not easy to draw a different conclusion from the vague and undefinable sensations or movements, very varying in character, but all comprehended under the term *aura*—sensations of numbness, pain, tingling; feelings as of a current of cold vapor; movements of shuddering or spasm, beginning in a distant part, and traveling toward the head; for the most probable interpretation of these symptoms is, without doubt, that of Dr. Watson, namely, this—that they are in some degree analogous to the numb and tingling feelings which are the frequent precursors of paralysis and apoplexy, or to the globus of hysteria.

¶ 100. *The phenomena of "morbid irritability" are not at variance with the premises.*

What is "morbid irritability?" It is not inflammation: it is not fever: it is some undefinable and negative state which occurs frequently in teething, in worm disease, in uterine derangement, and in many other cases—a state in which the patient is unusually depressed by depressing influences, and unusually excited by exciting influences. But what is this state? Is it anything more than mere ex-

haustion? In difficult teething the strength is worn away by pain and want of sleep; in worm disease, the system is ill nourished, and in all probability ill fed also, and the claims of the parasites will not improve this condition; in uterine disorders, the health is very likely to be seriously undermined by pain and by sanguineous and other discharges. In each case there is unequivocal exhaustion of body and mind, and many of the signs of "morbid irritability" appear to be nothing but the necessary signs of such exhaustion. And for the signs of irritation which may be associated with the signs of "morbid irritability," there is no reason to suppose that they may not be accounted for sufficiently upon the view of irritation already offered (¶ 98).

¶ 101. *The condition of the respiration and circulation during convulsion is one which necessitates the conclusion, that the convulsion is connected with a state of depressed nervous energy, and not with a contrary state of things.*

In epilepsy and in epileptiform convulsion, the deathly pallor of the countenance, and the comparative or absolute pulselessness at the wrist, which usher in the paroxysm, and the signs of suffocation which are present in the paroxysm itself, show very plainly that the convulsion is connected with a state of things in which the supply of arterial blood to the brain and other nervous centers, small and great, must of necessity be arrested. They show that the conditions of the epileptic and epileptiform fit are not unlike the conditions of the fit which is brought about by hæmorrhage or suffocation, and about which

enough has been said already. Nay, they show that the condition of the fit in all these cases is essentially the same, and they make it possible to apply to the explanation of epilepsy and epileptiform convulsion all the conclusions which were formed when speaking of the convulsion connected with hæmorrhage and suffocation.

And certainly there is nothing in the history of convulsion in hysteria and chorea which can militate against this conclusion.

¶ 102. *The general conclusion to be deduced from the condition of the functions of respiration, circulation, and innervation in convulsion is this—that the pathology of convulsion is as much in harmony with the view of muscular motion set forth in these lectures, as it is at variance with the current view on the subject—that, in fact, convulsion is connected with a state of depressed vital energy, and not with a contrary state of things.*

All the previous considerations lead to this conclusion, and to this conclusion only; and if I have failed to make this plain it is now too late to make it plainer.

LECTURE VI.

The main conclusion at which I arrived in my last lecture was—that convulsion is connected with a state of depressed vital energy, and not with a contrary state of things.

I showed that the respiration is either altogether arrested or greatly embarrassed in every form of convulsion; and I argued that this state of the respiration must involve, as a matter of course, a corresponding degree of depression in every vital function.

I showed that every form of convulsion is ushered in by paleness of the countenance, by great feebleness of the pulse at the wrist, and by other signs of failure in the circulation; and I argued that this state of things must be incompatible with anything except extreme depression in every vital function.

I showed that the strong and full pulse which so often accompanies the fully-developed epileptic or epileptiform convulsion is a pulse of *black* blood, and not a pulse of *red* blood, the pulse of suffocation—the *apnœal* pulse, and not the pulse which owes its increased fullness and force to the increased injection of arterial blood into the vessel; and I argued that the pulse in question is quite consistent with the conclusion that convulsion is connected with vital depression, and not with vital excitement.

I strove, in fact, to expose the fallacy of the view which supposes that the pulse of the convulsive paroxysm is rendered full and strong by an increased injection of arterial blood into the vessel, and which would account for the convulsion by supposing that this increased injection of arterial blood has produced an increased development of nervous energy in some parts of the nervous system, in the medulla oblongata more especially.

I showed that convulsion is never coincident with a state of active febrile excitement of the circulation —that it is associated with the cold stage before the hot stage, or with the cold stage after the hot stage, and never with the hot stage itself, and I advanced some reasons for believing that the hot stage of fever is actually antagonistic to convulsion.

I showed that the convulsion which may attend upon the close of Bright's disease is connected with a pale and watery condition of the blood, with uræmic poisoning it may be, and with unmistakable signs of great vascular debility—with a state which is quite incompatible with vital energy.

I showed that convulsion must not be looked upon as the consequence of active "determination of blood to the head." I showed that the place of convulsion in connection with active inflammation of the brain or its membranes is along with rigor and pain in the cold stage before the hot stage, or along with subsultus in the cold stage after the hot stage, and not during the hot stage itself. I showed, also, that in the cases of apoplexy where partial convulsive movements are associated with symptoms of active determination of blood to the head, and with a

full pulse and hot skin generally, that the convulsion, which is clearly owing to the "irritation" arising from the lacerated or compressed brain-fiber, might, in all probability, be general instead of partial, if there had not been this state of vascular reaction in the head and elsewhere to counteract it.

I showed that the peculiar condition of the nervous system which is known under the name of *irritation*, and which in all cases has so much to do with the production of convulsion, is in no sense the equivalent of inflammation. I pointed out how the effect of irritation in the vaso-motor nerves is contraction in the vessels—a state the very opposite to that of inflammation. I pointed out how a continuance of this irritation in the vaso-motor nerves might eventually lead to dilatation of the vessels—the first step in the process of inflammation—by depriving the nerves of their irritability, and by thus leaving the muscular coats of the vessels at liberty to fall into that state of relaxation which is natural to muscle when left to itself. And thus I was able to account for the fact, that traces of inflammation should be sometimes absent and sometimes present in certain cases of convulsion after death, for all I had to do in order to this was to suppose that the state of irritation had not ended in inflammation in the former case—had not progressed unto the point in which the vaso-motor nerves had become paralyzed, and that it had ended in inflammation—had resulted in paralysis of the vaso-motor nerves, in the latter case. I was obliged, indeed, to look upon the state of irritation as connected with anæmia rather than hyperæmia—as the accompaniment of a state

involving vital depression, rather than the contrary —as an additional reason in favor of the conclusions already arrived at, and not as an objection to these conclusions. In a word, the general conclusion which I deduced from the consideration of the respiration, circulation, and innervation during convulsion was this—that convulsion is the sign of vital depression and not the sign of vital excitement—that the pathology of convulsion is as much in harmony with the view of muscular motion set forth in these lectures as it is at variance with the current view of muscular motion.

And further, I pointed out how the depressed vitality of the nervous system arising from a failure in the respiration and circulation, may give rise to a reversal in the electrical relations of the exterior and interior of the nerve-fibers in certain nerve-centers, especially in the medulla oblongata,—how this reversal may disturb the state of static electrical equilibrium (which is the state of the nervous system when this system is not in action), by giving rise to a succession of electric discharges analogous to those of the torpedo,—and how these electric discharges may, according to the physiological premises, issue in convulsion. I pointed out, in fact, that the convulsion may be connected with a definite and intelligible disturbance in the electricity of the nervous system, and that this disturbance may be traced to the failure in the respiration and circulation which has been seen to be associated with convulsion. The argument is too long and too complicated to admit of being restated in a few words; and, therefore, all I can do now is to express a hope

that what I have said upon this subject in the last and in the previous lectures has not altogether escaped the recollection of those who did me the honor to listen to me.

In my present lecture I have to make a few hasty remarks upon certain points which seem to be of primary importance in the treatment of convulsion.

§ II. On the therapeutics of convulsion.

¶ 103. *There is reason to believe that the diet in many cases of chronic convulsive disorder ought to contain somewhat more than an average quantity of oily and fatty matters, and somewhat less than an average quantity of lean meat.*

There is a common notion, not confined to non-medical circles, that lean butcher's meat is the one thing necessary to strengthen a weak system; and I believe that the carrying out of this notion not unfrequently complicates the difficulties which prevent the successful treatment of the cases under consideration, and of many other cases also. With such a diet, as it seems to me, the blood is likely to get into a semi-gouty condition unless there be a degree of activity in the circulation and respiration which is not likely to be met with in epilepsy, chorea, hysteria, or any other form of chronic convulsive disorder; and I have an impression that the undoubtedly beneficial influence of bromide of potassium in so many cases of epilepsy is owing, in part at least, to the fact that this salt, like iodide of potassium, corrects the semi-gouty condition of the blood arising from this and other causes. At any rate, I have no doubt as to the practical advantages

of so regulating the diet in the cases under consideration as to diminish the usual allowance of fibrinous articles, and to increase the usual allowance of oily and fatty articles. I have increased the quantity of the latter articles for the same reason as that which has led me to employ cod-liver oil in many of these cases, and of which I shall have to speak presently.

¶ 104. *There is reason to believe that suitable gymnastic exercises are very beneficial in many chronic convulsive cases.*

During the last three or four years I have seen several cases of epilepsy, chorea, and hysteria, in which undoubted good has resulted from the adoption of a regular course of suitable gymnastic exercises; and the more I see the more I am satisfied that a course of this kind is a very important adjuvant in the treatment of these and many other cases. I can even call to mind more than one case of epilepsy in which the patient has said that he has warded off an attack which seemed to be imminent by a bout at the trapezium; and I have at present a case under treatment in which good seems to have been done by adopting a practice recommended by Dr. Henry Silvester in the treatment of consumption* —a practice which may perhaps be brought under the head of gymnastics. Having ascertained that the mere dead weight of the arms has the effect of reducing the amount of air which can be taken into the chest to the extent of ten cubic inches or thereabouts, Dr. Silvester proposes that a phthisical

* "The Physiological Method of Treating Consumption." Churchill, 1862.

person shall now and then eke out his insufficient respiration by breathing in such a manner as to get rid of this weight—by breathing, that is to say, with the hands taking hold of something fixed at a sufficiently high level, or, what answers the purpose still more easily, with the hands clasped together and resting upon the top of the head. And this proposition appears to have much to recommend it, not only in phthisis, but also in other cases in which, as in epilepsy, the respiration is wanting in activity. I also think that a collateral argument in favor of gymnastics may be derived from Dr. Silvester's investigations upon artificial respiration; for these investigations show that as much as from nine to forty-four cubic inches of air may be made to pass in and out of the chest by merely pulling the arms upward and then bringing them back to the sides, and that this movement of the arms is in itself a mode of performing artificial respiration which is more effectual than any other. Of course, the beneficial influence of gymnastics is not confined to the respiratory function. On the contrary, this influence tells equally upon the circulatory and upon all other functions, as it indeed must do if it act in this manner upon the respiration, for the activity of the respiration is a fair criterion of vital action generally.

¶ 105. *There is reason to believe that more harm than good may be done by the frequent use of purgatives in many forms of epileptic and other convulsive disorders.*

Abernethy said, "purging medicines sometimes

relieve unpleasant sensations; but they do not in general produce even this effect, and all active purges seem to me to increase disorder."* This is saying much; but, judging from my own experience in the matter, it is not saying more than is fully borne out by the facts of the case. It is, no doubt, of great moment to prevent the accumulation of effete matters in the bowels, and to remove such accumulation when it exists; but whether purgatives are the best means at our disposal for this purpose is another question. If the bowels do not act with sufficient regularity, there is, in all probability, some error in the diet—some excess of lean meat, some deficiency of fatty and oily matter, or of culinary vegetables and fruit; and the first thing to be done is, obviously, to correct this error. And this is often all that will be wanted, if care be taken to explain to the patient that the bowels can act without purgatives, and that he need not, particularly if he is advanced in life, be altogether cast down, if now and then they do not act every day. Indeed, if the diet be properly looked to, and if this explanation be made, the patient will generally have the satisfaction of finding his tongue clean when he happens to look at it, and of forgetting his stomach and bowels altogether, except at the times when he ought to remember them. At any rate, so far as my own experience goes, I am quite satisfied that the great majority of persons suffering from "head-symptoms" in various forms are better without purgatives; and this the more if

* "On the Constitutional Origin and Treatment of Local Diseases." 8vo. London, 1827, p. 89.

they had previously been in the habit of using them regularly.

It may be doubted, also, whether the common practice in this country of choosing mercurial purges is altogether sound and good. It is supposed that the mercury favors the elimination of bile; but this supposition may be challenged by any person who is disposed to be captious or skeptical. Professor Kölliker tried the effects of calomel upon the secretion of bile in dogs with biliary fistulæ; but it is difficult to rest any positive conclusions upon his experiments. Once the bile was increased by the action of the calomel, twice it seemed to be diminished. Since this time, Dr. George Scott,* formerly one of the physicians to the British Hospital at Renkioi during the Crimean war, has entered upon the same inquiry; and the conclusion to be based upon his experiments is sufficiently definite, namely—that there is a diminution in the amount of fluid bile and bile-solids secreted after the administration of large doses (purgative doses) of calomel. Dr. Scott's experiments are four in number, dogs being the creatures victimized; they were performed before several witnesses in Dr. Beale's laboratory; and it is only right to say that they were performed with every necessary precaution to avoid error. Nor must I omit to mention, as bearing upon the same point, that there is some reason to suppose that the boiled spinach or calomel stools of children owe their peculiar characters, not to the presence of altered bile, but to the presence of blood, effused apparently from the mucous mem-

* Beale's "Archives of Medicine," No. 3. 1858.

brane, and altered by the action of sub-sulphide of mercury.

I drop these remarks in passing without laying any stress upon them: and, at the same time, I take leave of the subject of the present paragraph, for I have nothing new to say upon the relative advantages or disadvantages of one aperient or purgative as compared with another, or of aperient or purgative draughts as compared with aperient or purgative enemas.

¶ 106. *There is reason to believe that bromide of potassium is an invaluable remedy in many cases of epileptic and epileptiform disorder.*

At a meeting of the Royal Medico-Chirurgical Society in the spring of 1853, Sir Charles Locock, then President of the Society, in some comments upon a paper which had just been read, said,—"About fourteen months ago I was applied to by the parents of a lady who had hysterical epilepsy for nine years, and had tried *all* the remedies that could be thought of by various medical men (myself among the number) without effect. This patient began to take *bromide of potassium* last March twelvemonth, having just passed one of her menstrual periods, in which she had two attacks. She took ten grains three times a day for three months; then the same dose for a fortnight previous to each menstrual period; and for the last three or four months she has taken them for only a week before menstruation. The result has been, that she has not had an attack during the whole of the period. I have tried the remedy in fourteen or fifteen cases,

and it has only failed in one; and in that one the patient had fits, not only at the time of menstruation, but also in the intervals."* Sir Charles also said that there was great uterine irritation in all these cases, and that his object in using the bromide was to correct this state of disorder.

In the course of the four or five years following, I put this mode of treatment into practice in five or six cases of epilepsy in which uterine irritation was a prominent feature, and with very satisfactory results upon the whole. In two of the cases, indeed, fits which had occurred at the rate of two or three a month for the greater part of two years, remained in abeyance so long as the treatment was persevered in, and returned more than once when the patient got tired and left off the medicine.

In the summer of 1858 I began to give this medicine almost promiscuously in cases of epilepsy and epileptiform disorder, and from that time to this I have been continually finding fresh reasons for persevering in the practice. I have given it in cases the most dissimilar in their character—almost promiscuously, as I have said; and the conclusion at which I have arrived is, that bromide of potassium is the only remedy in epilepsy upon which most dependence can be placed, and that a brighter future in the fortunes of epileptics may be dated from the evening when Sir Charles Locock gave utterance to the words which have just been quoted.

The physiological effects of bromide of potassium require further investigation. "M. Huette states that this compound possesses narcotic and anæsthetic

* "Medical Times and Gazette," May 23, 1853.

powers of a very peculiar and energetic kind, if from three to four ounces, in doses gradually increased from ten to twenty scruples, be taken within a period of fifteen days. A dull headache is the first effect; stupor and drowsiness soon follow. This is interrupted by delirium, resembling the incoherence of idiotcy, mingled with hallucinations. The muscular strength rapidly gives way, and with it the general sensibility. This latter effect, however, is very seldom carried to any considerable degree, and the cases in which the bromide causes sufficient insensibility to admit of surgical operations being performed are rare. It cannot, therefore, replace ether or chloroform. The symptoms above described continue as long as the use of the medicine; but the functions of organic life are not disturbed, and the effects rapidly subside under the use of purgatives. One effect is peculiar:— that even in small doses it rapidly and completely annihilates the sensibility of the pharynx and velum palati, to such an extent that these parts may be tickled without exciting the least effort at deglutition."*

This latter effect is not unfrequently produced by the doses necessary to produce a favorable impression in cases of epilepsy, by doses, say, of fifteen grains, three times a day, and therefore it is possible that it may be necessary to assume some specific action upon the medulla oblongata, or upon the nerves belonging to this center, in order to explain the *modus operandi* of the bromide. In other cases, however, there is no such effect, and in no case can it be said to be necessary to push the dose far enough

* "Pereira's Mat. Med.," vol. i. p. 526.

to produce it. It is, indeed, no easy matter to explain the *modus operandi* of the bromide. Is its action analogous to iodide of potassium? Does it correct errors of assimilation arising from overeating or overdrinking? Does it keep the blood in the state of purity necessary to the proper discharge of its manifold offices in the economy? Certain it is that many epileptics are continually gorging themselves with food, and that a medicine is not unlikely to do them good which can act upon the system in the same way as that in which iodide of potassium is found to act in relieving a gouty condition. Certain it is that a patient feels brighter and better under its proper use, and not less certain that in many cases its action appears to be greatly facilitated by the addition of a small quantity of iodide of potassium or bicarbonate of potass. Nay, I can say this—that before beginning to use the bromide of potassium I had found decided benefit in many cases of epilepsy from occasional doses of bicarbonate of potass, or sesquicarbonate of ammonia, with or without a little iodide of potassium, or tincture of colchicum, or wine of white hellebore. In a word, there appear to be sundry good reasons for believing that the *modus operandi* of bromide of potassium is, in part at least, by an alterative action upon the blood—an action in some degree analogous to that of iodide of potassium.

Bromide of ammonium, so far as my experience goes, appears to act in all essential respects like bromide of potassium. Bromide of iron, on the other hand, appears to act differently. At any rate, I have notes of about thirty cases of epilepsy, in which I

tried bromide of iron and bromide of potassium month by month alternately for some time, and in which, as a rule, the patient was decidedly better while taking the latter compound. Of the action of bromine alone I have no experience.

¶ 107. *There is reason to believe that the action of cod-liver oil is very beneficial in many cases of chronic convulsive disorder.*

For the last four years I have employed cod-liver oil in many cases of epilepsy, chronic epileptiform disorder, chorea, and hysteria; and, so far as I can judge, I have no reason to be dissatisfied with the results. I can also refer to the experience of my friend and colleague, Dr. Anstie, as bearing out my own experience in this respect most fully. I was led to this practice, and also to that of recommending a fair amount of fatty and oily articles of diet, by remembering that fatty matter is, as is seen in the following analysis of human brain by M. L'Herétier, an important ingredient of brain-tissue.

	Infants.	Youths.	Adults.	Aged persons.	Idiots.
Fat	3·45	5·30	6·10	4·32	5·
Phosphorus . .	·80	1·65	1·80	1·09	0·85
Albumen . .	7·	10·20	9·40	8·65	8·40
Osmazone and Salts .	5·96	8·59	10·19	12·18	14·82
Water . . .	82·79	74·26	72·51	73·76	70·93

Remembering this fact, and remembering also that the composition of nerve-tissue is substantially the

same in all parts of the nervous system, I came to the conclusion that fatty matter might be as essential to the proper nutrition of nerve, as flesh meat is to the proper nutrition of muscle. Remembering the reasons which oblige me to believe that the function of innervation is carried on very imperfectly in all convulsive maladies, and that a weak and starved nervous system may be supposed to have to do with this imperfect innervation in some cases, I came to the conclusion that a disposition to convulsion might be an additional reason for the use of fatty matter, either in the form of medicine or food: and, be this theory right or wrong, I think, as I have said, that I have no reason to be dissatisfied with the results of putting it in practice.

¶ 108. *There is reason to believe that phosphorus is a suitable remedy in many cases of chronic convulsive disorder.*

For the last four years I have used phosphorus in the majority of the cases of convulsion in which I have used cod-liver oil, and for the same reasons and with the same results. I asked myself whether the fact set forth in the preceding table (¶ 107), that phosphorus is present in nerve-tissue, and that the amount of this ingredient seems to have some direct relation to the activity of the nervous function, being as much as two per cent. in adult life, and below one per cent. in infants and idiots, might not show that phosphorus is required as food by a weak nervous system,—as much required, perhaps, as iron in cases where there is a deficiency of red corpuscles in the blood: and this question, once put, seemed

to require an answer in the affirmative. "In small doses," says Dr. Pereira, "phosphorus excites the nervous, vascular, and secretory organs. It creates an agreeable feeling of warmth in the epigastrium, increases the fullness and frequency of the pulse, augments the heat of the skin, heightens the mental activity and the muscular powers, and operates as a powerful sudorific and diuretic." In large doses, phosphorus, without doubt, is a caustic poison; in proper doses it produces the very changes which are necessary in epilepsy and in other cases of chronic convulsive disorder. In proper doses, and under the eye of a medical man, it is quite innocent of harm, and it may be productive of much good. This inference is that which may be drawn from what I have said; and this inference, so far as I can see, is not contradicted by experience. Given in the large doses in which phosphorus has been given in a few cases already on record, the good resulting may have been doubtful—very doubtful; but this experience is nothing to the point, for there is no reasoning in any case as to the effects of medicinal doses from the effects of poisonous doses. Given in medicinal doses, I have seen enough to know that this remedy may be given, not only without harm, but with the unmistakable promise of real and substantial good. The form in which I first gave the phosphorus was the phosphorated oil of the Prussian Pharmacopœia, a preparation which is made by dissolving twelve grains of phosphorus in a fluid ounce of almond oil by the aid of warm water. About four grains of the phosphorus is taken up, and the usual dose is from five to ten minims. I gave this

oil along with cod-liver oil in a little orange wine, twice or thrice a day. In many cases, however, this mixture proved to be so nauseous that the stomach refused to tolerate it; and lately I have often given the oil and the phosphorus separately, using the ethereal tincture of phosphorus of the French Codex as the vehicle for the phosphorus. I have given the oil with tolerable regularity as long as it seemed to be wanted, and the tincture now and then, especially when the symptoms called for a stimulant. I direct one fluid drachm of the ethereal tincture to be mixed with two fluid ounces of sulphuric ether, preserved in a capped bottle, the dose being half a fluid drachm to one fluid drachm, mixed with water at the instant of swallowing it. In the ethereal tincture of phosphorus of the French Codex, four grains of phosphorus are dissolved in one fluid ounce of ether, and consequently the strength is the same as that of the phosphorated oil of the Prussian Pharmacopœia.*

* Since this lecture was delivered, I have used the hypophosphite of soda, magnesia, or lime, as a means of administering phosphorus, and the result is that I have almost entirely discarded the two preparations mentioned in the text. Dr. Churchill, of Paris, considers these hypophosphites as specifics in phthisis: he supposes that the system is in want of phosphorus in this complaint, and that these salts supply this want: and what he says on the subject led me to think that the hypophosphites might be an excellent form in which to give phosphorus in *any* case where this substance was wanted. The hypophosphites, it must be understood, are altogether different from the phosphates. In the phosphoric acid of the phosphates the phosphorus is held a fast prisoner by very strong affinities—by affinities which are not likely to be overcome by any power of decomposition belonging to the animal economy: in the hypophosphorous acid of the hypophosphites, on the other hand, the phosphorus is in such loose combination as to be at liberty to take fire if held near a flame.

¶ 109. *There is reason to doubt the suitableness of belladonna as a remedy in many cases of epilepsy and other forms of chronic convulsive disorder.*

There is reason for believing that the dimensions of the iris in the state of rest are in great measure dependent upon the condition of its vessels as to fullness or emptiness, the iris being broad and the pupil small when this condition is one of hyperæmia, the iris being narrow and the pupil large in the opposite case of anæmia. When the superficial and deep-seated parts of a lateral half of the head are made hyperæmic by dividing the sympathetic nerve in the neck, the iris on that side is broad and the pupil small; when this state of hyperæmia is made to give place to a state of anæmia by exposing the distal portion of the trunk of the divided sympathetic to the shocks of a coil-machine, the iris at once becomes narrow and the pupil large. When the vessels of the head are made turgid by hanging an animal with the head downward, the iris is broad and the pupil small; when the vessels of the head are emptied by the local application of ice, by hæmorrhage, or in any other way, the iris becomes narrow and the pupil large. When symptoms denoting active determination of blood to the brain are present, as in the hot stage of acute meningitis, the iris is broad and the pupil small; when the head-symptoms are indicative of an anæmic rather than of a hyperæmic condition of the brain, as in hydroencephaloid disease, the iris is narrow and the pupil large. There are, indeed, good reasons for believing that the broadness or narrowness of the iris during the state of rest, and the consequent

smallness or largeness of the pupil, are in some degree dependent upon the fullness or emptiness of the vessels of the iris; and the same reasons are also valid for the purpose of showing that the vascular fullness or emptiness of the iris may be taken as the index of the fullness or emptiness of the vessels of the brain. There are reasons for believing, that is to say, that a narrow iris and a large pupil must indicate an anæmic condition of the brain, and that a broad iris and a small pupil must point to a contrary condition of hyperæmia in these vessels; and these reasons have led me to think that belladonna, which has the power of making the iris narrow and the pupil large, must, in order to be useful, be given in cases where the iris is broad and the pupil small, and where, consequently, a hyperæmic condition of the brain is the disorder to be combated, and not in cases where, as in epilepsy and in convulsive disorders generally, the evidence goes to show that the vascular condition of the brain is much more akin to anæmia than to hyperæmia. And, certainly, I have yet to learn that the good resulting from the empirical employment of belladonna in epilepsy, and in disorders analogous to epilepsy, is so unequivocal as to require me to disregard this theoretical objection. Nay, I may say this—that I know of more than one practitioner of large experience who has come to the conclusion that belladonna is more likely to do harm than good in these cases, if it be pushed far enough to produce even a shadow of its physiological action upon the system.

¶ 110. *There is reason to believe that opium may be a more suitable remedy than belladonna in some cases of epilepsy and in some other forms of convulsive disorder.*

Opium differs from belladonna in causing contraction of the pupil instead of dilatation; and therefore, for the reasons set forth in the last paragraph, opium differs from belladonna in producing a hyperæmic instead of an anæmic condition of the brain, and in being suitable in cases in which belladonna is not suitable—in cases where the brain is anæmic rather than hyperæmic—in some cases, it may be, of epilepsy among others. As yet, however, I have met with few cases in which I have thought it expedient to test the correctness of this theory by putting it in practice.

¶ 111. *There is reason to doubt the efficacy of zinc as a remedy in ordinary epilepsy, and in cases akin to ordinary epilepsy.*

I am disposed to look upon zinc as having an action upon the system which is directly opposed to that of iron. Iron, as all know, has an astonishing power of favoring the nutrition and multiplication of the blood-corpuscles. Zinc, on the other hand, blanches the system, and induces, before long, the state which was once known as *tabes sicca*, and for which no other name has yet been devised. Zinc, indeed, exercises a peculiar desiccating influence upon the system. Dr. Pereira mentions the case of an epileptic gentleman who took daily, upon an average, twenty grains of oxide of zinc until he had

swallowed 3246 grains, and who at the end of this time was pale in the extreme, sallow, wasted away, almost idiotical, with the tongue thickly coated, the bowels constipated, the inferior limbs cold and œdematous, the abdomen tumid, the arms cold and shriveled, the skin dry and almost like parchment, the pulse slow, thready, and scarcely perceptible. This patient experienced no change for the better in his fits; but he soon recovered from the effects of the zinc under appropriate treatment. I have also myself seen four cases, different from this one only in being not quite so extreme; and I have met with many cases in which the prolonged use of zinc, in one form or another, has produced decided sallowness and bloodlessness of the complexion. I find also that brass-founders, who are exposed to the fumes of deflagrating zinc, are often dried up and wizened in a curious manner. And Dr. Greenhow has recently shown, in addition, that these men are apt to suffer, particularly in the afternoons of the days spent in the casting-shop, from what is called "brass-founders' ague"—a disease beginning with malaise, tightness or constriction in the chest, nausea, and repeated rigors, and ending in profuse sweating after a short and faintly-marked hot stage. Here, then, is evidence that zinc is capable of producing a form of convulsive malady—for rigor is a form of such malady—as well as of producing the *tabes sicca* which has been described. Here, indeed, is evidence which may perhaps throw some light upon the disorders of the nervous system in which zinc is likely to do good or harm. That zinc does not always do good in these disorders is evident.

My friend and colleague, Dr. Marcet, who has for some time past given oxide of zinc very extensively in these disorders, reports favorably of the result in some cases, but not in others. He does not report very favorably of this mode of treatment in ordinary cases of epilepsy; and his report, I am satisfied, will not clash with the experience of the great majority of practitioners. Nay, it is a significant fact that M. Herpin, who has written a thick volume* in praise of the virtues of oxide of zinc in epilepsy, has for some time been dissatisfied with his own favorite remedy, even to the extent of discarding it not unfrequently for a remedy which savors more strongly of the materia medica of the middle ages than that of the nineteenth century—poudre de Neufchâtel, or, in plain words, powder of fried mole. Dr. Marcet reports more favorably of the action of oxide of zinc in those cases in which he had to deal with various vague head-symptoms without convulsion—a result which will also coincide with the experience of not a few. How, then, is this? Is it that the zinc does good in those cases of brain disorder in which there is a disposition to congestion of the brain, without deficiency in the amount and quality of the blood, by virtue of that power which produces *tabes sicca* when pushed to an injurious extent? Is it that the zinc does good when the condition to be combated is hyperæmic, and harm when this condition is anæmic? These are questions to which I know no better answer than that which is contained in the preceding considera-

* "Du Prognostic et du Traitement curatif de l'Epilepsie." 8 vo. Paris, 1852.

tions; and this answer is so plain as to require no further comment to make it plainer.

¶ 112. *There is reason to believe that alcoholic stimulants are very trustworthy antispasmodics in the prevention and treatment of convulsion.*

The wider experience of the last four years has not shaken my early convictions upon this point. On the contrary, I do not remember any one case in which there was not something to strengthen these convictions. I have very recently seen a case of aggravated chorea in which there had been no sleep for five days and nights, and no cessation to the movements of any moment, in which a wine-glass of port wine given every half hour, with an egg beaten up in the alternate doses, produced quiet and sleep in ten hours, and in which a continuance of the same treatment, only in a less vigorous style, left the patient well, so far as the chorea was concerned, in a week; and I could cite other cases, at least three, to the same effect. I could also cite many cases in which epileptic and hysteric convulsion was often averted by means of a proper use of alcoholic stimulants.

¶ 113. *There is reason to believe that blood-letting, in one form or another, may be permitted in certain cases of convulsion in order to prevent certain consequences of the convulsion.*

There is nothing in the pathology of convulsion to justify the notion that convulsion is likely to be prevented by blood-letting; but it is not difficult to understand that cases of epileptic or epileptiform

convulsion may be met with in which the veins of the brain may be so gorged with black blood as to put the patient in imminent danger of apoplexy, and in which this danger may be somewhat lessened by abstracting a small quantity of blood. And possibly this may be no wrong practice in such a case. At present it is plain that past experience is too much disregarded in this respect. Why, it may well be asked, if moderate blood-letting be so serious a matter, should a woman require periodical bleeding to keep her in health? Nor must the astonishing power of multiplication belonging to the blood-corpuscles be lost sight of in this matter. Speaking of the rapidity with which an anæmic patient became convalescent in St. Mary's Hospital, Dr. T. King Chambers says—and what he says requires no comment—"She weighs 8 stone, or 1792 oz.; of this $\frac{2}{7}$, or 512 oz., is blood; and of this blood $\frac{1}{10}\frac{3}{0}\frac{3}{0}$, or 60 oz., should be red globules. Now, the analyses of MM. Andral and Gavarret show that in cases of anæmia of at all a marked character (as this was) we may expect at least three-fourths of the red blood-disks to disappear; so that when she came into the hospital it may fairly be assumed that she did not possess more than 15 oz.; and now I think she may be assumed with equal fairness to have got up to 45 oz., which is conceding that she still wants a quarter of perfect health. By this she must have made 20 oz. of red blood-disks—that is the most important organic constituent of upwards of 150 oz. of blood—in a month!"*

* "Medical Times and Gazette," 11th January, 1862.

¶ 114. *There is reason to believe that the therapeutics of convulsion must be based upon the notion that vital power has to be reinforced, and not upon the contrary notion.*

This is the conclusion which may be based upon the physiological and pathological premises, and which is corroborated by what has been said in the present lecture. And this is the conclusion which, as I have to some extent shown elsewhere,* is in no sense contradicted by the therapeutical evidence which I would gladly have dwelt upon at greater length if time had permitted me to do so.

* "Epileptic and other Convulsive Affections of the Nervous System, their Pathology and Treatment. 3d Edition (incorporating the Gulstonian Lectures for 1860)." Post 8vo. London, Churchill, 1861.

LECTURE VII.

I occupied the hour allotted to me in my last lecture in making some desultory remarks upon the therapeutics of epilepsy and other forms of convulsion; and I came to the conclusion that the means to be employed with most likelihood of benefit are those which are calculated to recruit vital power in general and nerve-power in particular, and not those which have an opposite effect. I propose to occupy the coming hour in making some general remarks upon the pathology and therapeutics of tremor, and upon the pathology and therapeutics of spasm.

IV. ON TREMOR.

I propose to seek the information of which I am now in need in the history of common trembling, paralysis agitans, and delirium tremens, in the history of the rigors and subsultus of fevers, and in the history of the shakings of slow mercurial poisoning.

§ I. ON THE PATHOLOGY OF TREMOR.

(1) On the pathology of tremor as deduced from the condition of the respiration in this disorder.

¶ 115. *The condition of the respiration in tremor is one which warrants the belief that this disorder is connected with depressed and not with exalted vital power.*

The respiration is carried on very imperfectly in all forms of tremulous disorder. This is evident in the want of vital warmth, as well as in the comparatively small amount of air which passes in and out of the chest during a bout of trembling: and this also is what may be inferred from the depressed state of the circulation, of which something has to be said in the next section.

(2) On the pathology of tremor as deduced from the condition of the circulation in this disorder.

¶ 116. *The condition of the circulation during tremor is one of unmistakable depression.*

In an attack of common trembling the circulation is greatly depressed, and the pulse does not recover itself until the paroxysm is over; and in paralysis agitans the paleness and chilliness of the surface of the body, and the decided relief afforded by wine, tell a similar story. In delirium tremens, the cold perspirations, the quick and fluttering pulse, the moist and creamy tongue, are all significant facts. The initial rigor of fever, moreover, is coincident with wanting warmth, miserable pulse, sunken countenance, blueness of nails, *cutis anserina*, and other signs of vascular collapse, and subsultus with the most utter prostration of the powers of the circulation. And in mercurial tremor an inference as to the real state of the circulation may be drawn from the fact that the subjects of this disorder are not unfrequently in the habit of resorting to gin and other stimulants for the purpose of making themselves steady.

¶ 117. *There appears to be something uncongenial between tremor and an excited state of the circulation.*

The state of the circulation in the delirium of which trembling is the distinctive feature—delirium tremens, is quite different to the state of the circulation in the delirium in which there is no trembling. In the latter case—in the delirium of acute meningitis, for example—the skin, especially the skin of the head, is hot and dry, not cold and damp; the pulse is hard and strong, not weak and fluttering; the tongue is parched and dry, not moist and creamy — the condition, in short, is one of high fever, and not one which, as in delirium tremens, is more akin to collapse than to high fever. And it is not less certainly a fact that delirium tremens loses its characteristic trembling if acute head-symptoms and high fever make their appearance in the course of the disorder. Moreover, it must be borne in mind, as pointing to the same conclusion, that the initial rigors of fever disappear *pari passu* with the establishment of the vascular reaction of the hot stage, and that they return in the form of subsultus when this state of reaction has died out, and left the patient utterly prostrate and helpless. In a word, there are certain facts which appear to show that there is something uncongenial between tremor and an excited condition of the circulation.

¶ 118. *The condition of the circulation in tremor is one which warrants the belief that this disorder is connected with depressed and not with exalted vital energy.*

This is the only conclusion which can be formed

from the facts which have been under consideration hitherto, if only it be allowed, as it must needs be, that a due supply of arterial blood to the circulation is necessary to the due manifestation of vital energy in the system.

(3) On the pathology of tremor as deduced from the condition of the innervation in this disorder.

¶ 119. *The condition of the brain during trembling is one of unmistakable functional depression.*

The subjects of common tremulousness have a certain delicacy of constitution which cannot be overlooked; and, if not women, they have very generally a feminine habit of body and mind. It is also evident that they are altogether *unnerved* during the paroxysm, and that their thoughts and words are as little under control as their muscles. In old age and in paralysis agitans, every mental faculty has given way under the wear and tear of life; and during an actual bout of trembling, the mind loses for the time the command of the small stock of vital energy which is not yet expended. In delirium tremens, the mental state is passive in every point of view. The mind is confused, irritable, despondent, anxious, and tortured with gloomy forebodings or spectral delusions. Everything and everybody are objects of mistrust, or fear, or dread. In the initial rigors of fever, the mental state is one of dejection, languor, stupor; in subsultus, it is one of wandering silliness or of apathetic drowsiness. In slow murcurial poisoning, the failure of the mental powers keeps pace with the failure of the bodily powers, and the condition is

one of premature old age. In every case, in fact, the manifestation of brain-power is all but absolutely suspended during the act of trembling.

¶ 120. *There seems to be something uncongenial between tremor and an excited condition of brain.*

It is a common thing for a person to cease to tremble—to become steady—when he rouses himself, or when he is roused by others. It is a common thing for a patient who has been for days, or even for a longer time, in a state closely akin to that of delirium tremens, to lose all signs of tremulousness when the symptoms of the disease take the form belonging to acute mania. Nor would it be difficult to multiply instances to the same effect indefinitely. It would not be difficult to do this, but it is not necessary, for the statement which forms the heading of this paragraph is little more than a common truism.

¶ 121. *The condition of the innervation generally during tremor is one which warrants the belief that this disorder is connected with depressed and not with exalted nervous energy.*

In the different forms of tremor, the condition of the nervous system, as reflected in the state of the mind, has been seen to be one of weakness rather than strength. Nor is it possible to suppose that the condition of the cerebral hemispheres is different from that of other parts of the nervous system; for the condition of the respiration and circulation, which has just been described, is one which must necessitate a state of things in which the develop-

ment of nervous influence must be suspended, or all but suspended, not in one nerve-center only, but in all nerve-centers indifferently.

¶ 122. *The fact that tremor generally comes to an end during sleep is no objection to the conclusion that this disorder is associated with deficient nervous energy.*

If there be a connection between tremor and depressed nerve-power, it may seem, at first sight, that the trembling ought to be aggravated during sleep, when all brain-power and almost all nerve-power is more or less dormant; but this is not the conclusion which is arrived at after a few moments' reflection. For what is the state of the nervous system during sleep? It is, with the exception of a few centers here and there, a state of sleep. It is a state not remotely akin to paralysis. It is a state in which the muscles will become relaxed, and remain relaxed; for, upon either theory of muscular action, living muscles must become relaxed as soon as they are left to themselves, and must remain relaxed as long as they are left to themselves. And thus the fact that there is an end to tremor during sleep is no objection to the conclusion that this disorder is associated with a depressed condition of nerve-power in general, and of brain-power in particular.

¶ 123. *In tremor, therefore, as in convulsion, the condition of the three great functions of respiration, circulation, and innervation is one which warrants the conclusion that the morbid muscular contraction is the sign of depressed and not of exalted vital energy.*

After what has been said, this conclusion is obvious. After what has been said, indeed, I have left myself nothing further to say upon this subject.

§ II. ON THE THERAPEUTICS OF TREMOR.

¶ 124. *There is reason to believe that the means to be employed in the treatment of tremor are those which exalt vital tone in general and nervous tone in particular.*

The beneficial influence of stimulants in the treatment of many forms of trembling is a well-established fact; but the probability is that much yet remains to be learned upon this subject. I once knew, for example, a medical man who for some years was in the habit of giving a glass of strong and hot toddy before the onset of a paroxysm of ague, and who found that the sharp rigors of this disorder were almost always prevented, or at any rate greatly abbreviated and mitigated, by this means. It is also probable that much remains to be learned with respect to the beneficial influence of nutriment in many forms of tremulous disorder. Writing three years ago, I said,—"Within the last few months I have had four cases of delirium tremens in the Westminster Hospital, in which the treatment was by hot beef-tea and belladonna. A teacupful of very hot beef-tea, with bread sopped in it, was given regularly every hour, except during sleep; and every two or three hours, for the first couple of days, a draught containing ten drops of tincture of belladonna. The hot beef-tea was the only stimulant given. In each case the patient slept a good

deal during the first night, and the appetite for meat had returned on the fourth day. In each case the belladonna produced dilatation of the pupil and some dryness of the throat. This drug was given chiefly on the supposition that it might tend to counteract the dismal current of the thoughts (for, in full doses, as is well known, belladonna produces a gay and cheerful delirium), and this end may have been somewhat answered; but I am disposed to think that the part played by the drug was subordinate to that which was played by the hot beef-tea." And I am confirmed in this latter opinion by subsequent experience, for on treating several cases of delirium tremens simply with hot beef-tea and sop, the results were, to say the least, quite as satisfactory as those which were arrived at in the cases in which belladonna was used along with the hot beef-tea and sop. I may say also that much in all probability has to be learned with respect to the specially beneficial influence of the oily articles of food and medicine in the treatment of some kinds of trembling. I could cite, for instance, several cases of paralysis agitans in which the shakings were almost or altogether banished by the steady use of cod-liver oil and by introducing into the diet more butter and fat. In these cases, it is true, wine was given with a liberal hand; and wine, no doubt, did much good; but my impression is, that the lion's share of the credit belonged to the oily articles of food and medicine; and this also was the impression which the patient in each case formed for himself upon the matter. But I must not tax your patience, and waste the time which

yet remains to me, by entering into particulars, and for this reason, I will only say, that the rational treatment of tremor would seem to be that which avoids every cause of depression and exhaustion, which seeks after every means of increasing and establishing the strength, and which trusts to stimulants of one kind or another in any special emergency.

V. ON SPASM.

In order to gain the data from which to deduce the pathology and therapeutics of that form of convulsive disorder which is characterized by prolonged muscular contraction or spasm, I propose to glance, as hastily as I can, at the history of this disorder as it occurs in catalepsy, tetanus, cholera, hydrophobia, spasmodic ergotism, in certain diseases of the spinal cord, and in one or two spasmodic disorders of minor moment.

§ I. ON THE PATHOLOGY OF SPASM.

(1) On the pathology of spasm as deduced from the condition of the respiration in this disorder.

¶ 125. *There is reason to believe that spasm is associated with insufficient respiratory activity.*

In catalepsy the play of the lungs is almost or altogether imperceptible. In tetanus the breathing, never free, becomes more and more labored as the spasm gripes with firmer hold upon the respiratory muscles; and there are many moments in which the struggle for breath amounts well-nigh to mortal agony. In cholera the surface of the body is cold,

clammy, and blue, the respiration shallow and hampered, and the breath cold. In hydrophobia there is an abiding sense of suffocation, as from some impediment in the throat, and the breathings are hurried and often interrupted by sobs and sighs. In acute spinal meningitis and myelitis, dyspnœa is a prominent phenomenon, and the vital powers of the system soon succumb to want of breath. In laryngismus stridulus the spasm is accompanied by actual suffocation; and, in a lesser degree, so also in whooping-cough. In every form of spasm, indeed, the condition of the respiration is more or less obviously one which shows that this disorder is connected with vital exhaustion and not with vital excitement, if, as must needs be, the amount of respiratory action may be taken as a criterion of the amount of vital energy in the system at the time.

(2) On the pathology of spasm as deduced from the condition of the circulation in this disorder.

¶ 126. *There is reason to believe that spasm is associated frequently with a depressed state of the circulation.*

During the attack of catalepsy, the appearance of the patient is very like that of a corpse; and it may even be necessary to apply the ear to the chest to know of a certainty that the heart continues to beat. In tetanus, as all are agreed, there is no fever, and the pulse has no semblance of power except at those moments when dusky lips and other signs of deficient respiration are present to show that a fictitious power is being derived from the admission of imperfectly

aerated blood into the arteries (¶ 88). And in the tetanus arising from strychnia this conclusion acquires additional support from the fact discovered by Dr. Harley—that one action of this poison is to prevent the blood from becoming oxygenated. In cholera, the cramps are coincident with a state of almost pulseless collapse. In hydrophobia the condition of the circulation is the very opposite of true fever. In spasmodic ergotism, so far as we know, the pulse presents no sign of excitement throughout the whole course of the malady. And, certainly, a similar inference with respect to the state of the circulation is to be drawn from the seizures of cramp in the leg and elsewhere, which are so often met with in delicate and aged people, and particularly in persons in whom the period of old age is anticipated by white softening of the brain.

¶ 127. *There is reason to believe that spasm is antagonized rather than favored by an excited state of the circulation.*

In tetanus it appears to be the rule for the spasm to gain ground almost in exact proportion to the degree in which the pulse loses in true power. In hydrophobia it would seem as if the same law held good, for on analyzing the histories of a considerable number of cases, I find that there was less agitation, less convulsion, less spasm, where the circulation was less depressed than it is in the ordinary run of cases. Nor is a different conclusion to be drawn from the history of spasm as it is set forth in whooping-cough. For what is the fact? The fact is simply this—that the whoop, which is the audible sign of

the spasm, does not make its appearance until the febrile or catarrhal stage has passed off; that it disappears if pneumonia, bronchitis, or any other inflammation be developed in the course of the malady; and that it returns again when the inflammation has departed. Taken by itself this evidence, it is true, may not amount to much; taken in connection with what has gone before, and with what has still to come, it justifies the notion that spasm, like convulsion and tremor, is a disorder which is antagonized, rather than favored, by an excited condition of the circulation.

(3) On the pathology of spasm as deduced from the condition of the innervation in this disorder.

¶ 128. *There is reason to believe that spasm is associated with failure of brain-power.*

In the more severe forms of spasmodic disorder, the mental state during the spasm is one of abstraction, exhaustion, or prostration. In catalepsy the mind is either in a deep sleep, or else rapt in some absorbing vision. In tetanus the patient is alarmed, agitated, alive only to suffer. The cramps of cholera are attended by indifference to the future and by hopelessness, than which are no surer signs of mental prostration. In hydrophobia, the mind is in a state which may be said to be the exaggeration of that which is met with in delirium tremens. In spasmodic ergotism the state borders very closely upon fatuity. And in the minor forms of spasm, the evidence, so far as it goes, is to the same effect. Thus, for example, cramp in the calf of the leg is a common accompaniment of general or partial dementia,

and thus again, spasm in the stomach and bowels is not unfrequently the immediate result of sudden mental depression.

¶ 129. *There is reason to believe that spasm is antagonized rather than favored by inflammatory excitement in the nervous system.*

It is a common impression that spasm is in some especial manner a characteristic symptom of certain inflammatory conditions of the spinal cord, but it may be doubted whether this impression is justified by the facts.

In a certain number of cases of tetanus the spinal cord is found to exhibit post-mortem changes, and some of these changes point, without doubt, to the existence of recent inflammation. Thus, for example, in nineteen cases examined at Guy's Hospital, and referred to in an excellent report on tetanus by Mr. Poland,* the spinal cord was healthy in eight; firm, rigid, and of a pinkish hue in one; of natural firmness, but injected in one; injected in two; darker than natural in one; pinkish in two; of a "higher tint" in one; softened in one; and decomposed in one,—the last being a case which was fatal in four days, and in which the examination was made forty-one hours after death, and while the muscles were still perfectly rigid. In these nineteen cases, then, there is proof that inflammation of the cord is not essential to the existence of the tetanic symptoms, for the cord was healthy in no less than eight of the number. Nor can it be supposed that the cases in which the cord was healthy were the slighter

* "Guy's Hospital Reports." Third Series, vol. iii. 1857.

cases; on the contrary, it is certain that tetanus may occur in its most violent and acute form without leaving any traces of inflammation in the cord. I have myself been present at three post-mortem examinations in which this fact was as carefully demonstrated as anything of the kind could be demonstrated before the time when Mr. Lockhart Clarke had taught us to apply his beautiful method of examination to the detection of morbid changes in nerve-tissue.

Nor is it altogether certain that spasm is an absolutely essential accompaniment of primary myelitis, or primary spinal meningitis.

Acute spinal meningitis is often obscure enough at first, and this obscurity is generally increased by the presence of head-symptoms in one form or another, for, in the majority of cases, the spinal disease is only part of an affection in which the cranial nerve-centers are all in some degree implicated. Constipation, dysuria, or even retention of urine, are very early symptoms; indeed these, along with the malaise and feelings of weariness in the limbs which are so common to so many maladies, may be the first symptoms. As the malady gains ground, more decided symptoms make their appearance, and of these, morbid contraction in the muscles of the posterior part of the neck and trunk, pain along the course of the spine and shooting into the limbs, and morbid exaltation of the general sensibility are the most constant. The muscular contraction, as a rule, does not extend to the limbs, but it is confined, as I have just said, to the muscles of the posterior part of the back, and often to those of the back of the neck ex-

clusively. It varies from simple rigidity to tetanic spasm. It is not constant. It is manifested especially when the patient is moved, or in fear of being moved. It has been explained as an instinctive or semi-voluntary act of the muscles, of which the object is to fix the movable spinal vertebræ into an inflexible column, and in this manner to prevent the pain which the movement of these vertebræ would give rise to in the inflamed spinal cord; and this explanation would seem to have much to recommend it. At any rate, it is certain that these contractions are separated by intervals, and that these intervals are of very considerable duration if care be taken to keep the patient perfectly still. The pain along the spine and shooting into the limbs is generally very acute. It has, like its companion-symptom contraction, distinct remissions, and these remissions may be of considerable length, if the patient keep very quiet. It may exist independently of the contraction, but generally it is the precursor of the contraction. It is always exasperated by any movement. It is not increased by pressure, or by the application of a sponge wrung out of very hot or very cold water. Sometimes it does not shoot into the limbs. The morbid exaltation of general sensibility varies much, but it is always present in some degree, and generally in an extreme degree. In acute spinal meningitis in which the substance of the cord is free, the limbs are not paralyzed. They are wanting in power: their movements are fettered by the fear of the pain which any movement produces: but they are not paralyzed. In some cases, indeed, where delirium has masked the pain or suspended it, the patient has

continued to be able to move in bed, to stand, and even to walk about, until the very last. For the rest, it is only necessary to say that the skin is drenched in perspiration in the bouts of pain and contraction, that the respiration is panting and embarrassed, that the circulation is weak and excited, that the bladder continues paralyzed to the end, that the urine is not usually alkaline if care be taken to prevent it accumulating in the bladder, and that the bowels will sometimes recover the power of action which they had lost for awhile at the commencement of the malady. This is a free sketch of the history of acute spinal meningitis as it is presented in the cases and comments of M. Ollivier*—cases and comments which still supply by far the best information upon this subject.

In *acute myelitis* the primary symptoms are numbness and tingling of the fingers and toes, accompanied sometimes by difficulty of movement, and sometimes by feelings of cold in the same parts. Then these symptoms extend, first to the whole limbs, afterward to the greater part of the trunk. In a few instances, the malady has been ushered in by general or partial convulsion. In all cases, from the beginning, or soon after the beginning, there is some pain along the back; and in the great majority of cases this pain is increased by pressure, or by the application of a sponge wrung out of very hot or very cold water. The course of the malady is often very headlong. Rapidly the numbness and tingling give place to a state of complete anæsthesia, rapidly

* "Traité de la Moëlle 'Epinière et de ses Maladies." Par C. P. Ollivier (d'Angers). 2me ed., 2 tom. 8vo. Paris, 1827.

the difficulty of movement gives place to complete paralysis, rapidly grave lesions in nutrition, such as bed-sores, make their appearance—lesions which show that the vaso-motor nerves are participating in the same mischief as that which is deadening the common sensory and motor nerves. Now and then the sensory nerves may be affected without the motor; now and then the vaso-motor nerves may escape more or less; but generally all three sets of nerves are involved in the same ruin. In many cases, in all cases if the affection of the cord extend high enough, the sides of the chest are paralyzed, and the respiration is carried on almost exclusively by the diaphragm—a fact which causes dyspnœa to be a very prominent and distressing symptom of the malady. In many cases, also, the patient is much distressed by a feeling as of a tight girdle around the trunk at the line of junction between the paralyzed and non-paralyzed parts. In the end, the bladder is always paralyzed, and so is the sphincter ani, but not always from the beginning. And from the beginning to the end, the urine is usually strongly alkaline and offensive. These are the essential features of acute myelitis as they are sketched by the best authorities on the subject, especially by M. Ollivier; and, so far as my own limited experience enables me to judge, they are faithful to fact in all respects.

In acute myelitis, therefore, spasm in any form does not appear to figure as a symptom. In particular cases, no doubt, acute myelitis and acute spinal meningitis are associated in a manner which is more or less intimate, and therefore some spasm may not

unfrequently accompany the symptoms of myelitis; but, with ordinary attention to such evidence as that which is supplied by M. Ollivier, it is not difficult to come to the conclusion that spasm is not to be regarded as a symptom of pure acute myelitis. In acute spinal meningitis, on the other hand, spasm is looked upon, not only as a conspicuous symptom, but as the symptom which is especially characteristic. But this view of the matter is one which may be called in question, and which must be called in question. In the history which has been given, indeed, it has been seen, not only that there are remissions—often wide remissions—in the spasmodic accompaniments of acute spinal meningitis, but also that the contraction which is called spasm may be looked upon as an instinctive or semi-voluntary act of muscular contraction in the muscles of the spine, of which the object is to fix the spinal column immovably, and in this way to prevent the pain which the inflamed cord must experience at every movement of the column. It has been seen, in fact, that the contraction is scarcely to be called spasm at all. And, certainly, with the history of true spasm in tetanus to account for, it is difficult to look upon this contraction, even supposing it to be true spasm, as the characteristic symptom of inflammation of the meninges of the cord. For what does this history show? It shows that the signs of inflammation in the spinal cord, which are met with in certain cases of tetanus, are not to be regarded as essential to the existence of tetanic spasm; it shows that violent, constant, universal, involuntary, tetanic spasm is compatible with a perfectly non-inflammatory con-

dition of the cord; and in doing this, it makes it very difficult to believe that spasm is to be regarded as the characteristic symptom of an inflammatory condition of the cord. Nor is this difficulty lessened when it is remembered that the contractions of acute spinal meningitis are temporary and confined to the muscles of the spinal column—to the muscles of the cervical region of the spinal column, it may be, and that spasm is not a symptom of acute myelitis. Nay, with these facts to be taken into consideration, it is difficult to repress the thought that spasm may be antagonized rather than favored by inflammation of the spinal cord—that the place of spasm may be in the cold stage before the hot stage of this inflammation, in the stage of anæmia, and not in the stage of hyperæmia, in the stage of irritation, and not in the stage of inflammation, properly so called. And this difficulty becomes well-nigh insuperable when the history of spasm is taken in connection with what has been said in these lectures respecting the pathology of convulsion and tremor, and the physiology of muscular motion.

Nor are these conclusions at variance with the history of spasm as connected with chronic inflammation of the spinal cord or its membranes. The natural inference is that the history of the chronic forms of inflammation will be in harmony with the history of the acute forms: and I know of no *facts* which are in any degree calculated to set aside this inference. Indeed, I may say without hesitation, that so far as my own experience goes it is impossible to look upon spasm, or increased disposition to reflex movement, as a characteristic symptom of

chronic inflammation of the spinal cord or its membranes. My impression is that the cases of paraplegia in which spasm or convulsion in the legs is a marked phenomenon are those in which the disease has the effect of preventing the legs from receiving the "nervous influence" which they ought to receive continually from the great nerve-centers of the head and neck—that, in fact, this increased disposition to muscular contraction in these cases is analogous to that which is manifested in the hind legs of a frog or rabbit after the spinal cord has been cut across by a knife (¶ 43), and that the nature of the mischief which produces this interruption in the conducting powers of the cord is of no moment so far as the result in question is concerned, and this impression is, as I believe, fully justified by the facts.

But, it may be asked, is not the vagueness in the seat of the inflammation which may be developed in the course of various spasmodic disorders a certain proof that spasm is not to be regarded as a characteristic symptom of inflammation of the spinal cord, or of any other part of the nervous system? Undoubtedly there is this vagueness in the seat of the inflammation. In tetanus, for example, the traces of inflammatory action which may be met with are not in the spinal cord exclusively, but in various parts of the brain, in the nerves, and in other parts besides. And so also in hydrophobia. Thus, for example, in 46 cases of hydrophobia, of which the histories were carefully analyzed by my brother, Mr. John N. Radcliffe,* "the morbid appearances after death were found in the dura mater in 8, in the

* "Lancet," Sept. 1856.

arachnoid membrane in 10, in the pia mater in 16, in the velum interpositum in 2, in the choroid plexus in 12, in the cerebral hemispheres in 28, in the spinal cord and membranes in 18, in the medulla oblongata and pons Varolii in 4, in the tongue in 8, in the palate in 3, in the salivary glands in 2, in the pharynx in 19, in the œsophagus in 16, in the stomach in 20, in the intestines in 6, in the larnyx, trachea, and bronchial tubes in 31, in the ultimate ramifications of the air-passages in 24, in the heart in 4. These lesions consisted of every grade of injection of the blood-vessels, from the slightest blush to the most vivid, or dark, black congestion; of alteration in the consistency of the tissues, principally softening; of effusion of blood, and certain products of perverted nutrition and secretion. In several of the cases the lesions were of such a character that they have been classed with those resulting from common idiopathic inflammation; in a greater number of cases they were of that character which is found in the structural changes occurring in asthenic conditions of the system." Now this vagueness in the seat of these inflammatory and other structural changes I look upon as a very curious and significant fact—a fact which, perhaps, more clearly than any other single fact, is calculated to show the true relation of spasm to inflammation. It is calculated to show that inflammation of one particular nerve-center cannot be essential to the existence of the spasm. It is calculated to show that the cause of the inflammation may be as general as the cause of the febrile symptoms which are developed along with the inflammation—that, in

fact, the establishment of the febrile condition is only a first step in the establishment of the inflammatory condition, and that it is little more than an accident which fixes the seat of the inflammation in one nervous center rather than another, or in one part of the organism rather than in another. In the case of hydrophobia, indeed, it is calculated to put the inflammation which may be developed in the course of the malady in the position of a depurative process—a process which, like the inflammation developed along with the fever in the course of small-pox, is intended to rid the system of a morbid virus.

But I must bring those remarks to a close. I have wandered somewhat; but in the course of this wandering I have attained in some degree to the object I had in view, for I now find reason to believe that spasm, like convulsion and tremor, is related to an anæmic condition of the nervous system rather than to a hyperæmic condition,—that, in fact, spasm is antagonized rather than favored by a state of inflammatory excitement in the spinal cord, or in any other nerve-center. And further, I have found reason to believe that there is the same antagonistic relation between spasm and inflammation in any part of the body as that which I have shown to exist between convulsion and inflammation and between tremor and inflammation.

¶ 130. *There is reason to believe that all nerve-power is at a very low ebb during spasm.*

This reason is to be found in the depressed state of the respiration and circulation during spasm, of which sufficient evidence has been furnished already;

for it is to be supposed that this state of depression must involve a corresponding degree of failure in the development of nerve-power in every quarter where such development is provided for. Upon this point there can be no doubt whatever, for it is a law in physiology that the functional activity of an organ is directly proportionate to the supply of arterial blood to the organ.

¶ 131. *The condition of the three great functions of respiration, circulation, and innervation during spasm is one which warrants the conclusion that this form of muscular disorder is connected with depressed and not with exalted vital energy.*

This conclusion must follow, as a matter of course, from what has been said, and this only. Nor do I know of anything which has been left unsaid which is in any degree calculated to lead to a different conclusion.

¶ 132. *There is reason to believe that the key to the pathology of spasm, tremor, and convulsion is to be found in that view of the physiology of muscular motion which is set forth in these lectures, and that this view of the physiology of muscular motion is confirmed and established by the facts of pathology.*

All the previous considerations have gone to show that spasm, tremor, and convulsion are all alike dependent upon a defective development of vital power in general, and of nerve-power in particular. All these considerations, indeed, have proved to be as

much in harmony with the view of the physiology of muscular motion set forth in these lectures as they are at variance with the current view of the physiology of muscular motion—a view according to which the excess of muscular contraction is owing to a corresponding excess in the stimulation of a vital property of irritability in nerve and muscle. The physiology explains the pathology, and the pathology establishes the physiology. In pathology and in physiology, indeed, it appears to be one and the same story throughout.

§ II. ON THE THERAPEUTICS OF SPASM.

¶ 133. *There is reason to believe that the means to be employed in the treatment of spasm are those which are calculated to exalt vital energy in general, and nervous energy in particular.*

If the previous conclusions respecting the pathology of spasmodic disorders be correct, it is evident that antiphlogistic and sedative measures will hold no very prominent place in a sound system of therapeutics, and that the first and last indication of treatment will be to sustain and exalt every form of vital energy. In his remarks upon the treatment of tetanus, Dr. Watson says—"In all cases I should be more inclined to administer wine in large doses, and nutriment, than any particular drug;" and during the last few years several cases have been put on record in which the wisdom of this remark has been abundantly proved by successful practice. I have seen two cases in which strong and general tetanic spasms relaxed rapidly under a treatment in which the essential part was to give wine to a point just

short of inebriation. Nor do I see why a similar plan should not answer in hydrophobia if it were carried out promptly and decisively. I do not see that it might not be perfectly justifiable to try and save the patient by making him drunk as rapidly as possible. Of the good effects of wine and nourishment in the minor forms of spasmodic disorder, as laryngismus stridulus, spasmodic croup, and whooping-cough, I have no manner of doubt. I know, for example, of four cases where attacks of laryngismus stridulus which were frequently recurring under a treatment in which it was thought essential to "regulate the secretions" by gray powder and so on, were put a stop to at once and for all by a tonic and restorative plan of treatment—wine, beef-tea, steel, chloric ether, &c.; and I can speak with confidence of the beneficial results of a similar plan of treatment in a considerable number of cases of whooping-cough. Indeed, from what I have myself seen, I am strongly disposed to think that the unmanageableness of this last named disorder has mainly arisen from the fact that wine and nutriment have been withheld, or administered with a niggard hand; and for the rest, I will only say that the treatment of spasm, convulsion, and tremor, must be based upon one and the same general principle.

LECTURE VIII.

My last lecture was spent in investigating the theory and treatment of tremor and spasm, and the result of the inquiry was found to be in perfect harmony with the results previously arrived at respecting the theory and treatment of convulsion. My present lecture will be devoted to an examination of the theory and treatment of pain, and to the consideration of certain questions connected with paralysis.

VI. ON PAIN.

Pain is either of a neuralgic character, or it depends upon tenderness. The latter pain is, so to speak, accidental; it is only felt when a tender part is subjected to pressure, and it is generally associated with a congestive or inflammatory condition of the part. The former pain, on the other hand, may be spoken of as essential: it is more likely to be relieved by pressure than to be produced by it, and in the great majority of cases it is impossible to associate it with the faintest sign of congestion or inflammation in the painful part, or of feverish reaction in the system generally. It is possible, without doubt, to draw this broad line of distinction between these two forms of pain, but at the same time it is not to be denied that this line seems to be broken in some

parts, and that the two forms of pain interblend at these parts and lose their distinctive characteristics. How, then, is this? This is the question which I propose to answer; and this is a question which demands very grave attention, for, as I hope to show before I have done, conclusions of much theoretical and practical importance are at issue.

§ I. ON THE PATHOLOGY OF PAIN.

(1) On the pathology of pain as deduced from the condition of the circulation.

¶ 134. *Pain of a neuralgic character may be associated with a deeply depressed condition of the circulation.*

It is a well-established fact that neuralgia in its most excruciating form may occur again and again without either fever or inflammation. It is also a well-established fact that the majority of persons who suffer from neuralgia are of a feeble and excitable constitution, with the circulation in keeping with this state of things. Judging, also, from the pale and perspiring skin, and the miserable pulse, which are so generally met with in the actual paroxysm of neuralgia, it may be supposed that this paroxysm is associated with a state of the circulation in which the habitual depression is exaggerated. Indeed, the appearances during such a paroxysm are often calculated to remind one of the cold stage of ague, especially in that form of neuralgia which is met with in aguish districts, and in which malaria seems to figure conspicuously as a cause of the malady; for in this case the neuralgia is often obedient to the same law as ague so far as this—that it is

associated with rigors, that it begins and ends punctually at a given time, and that it is followed by an obscure hot fit. It would seem, indeed, as if the neuralgia and the rigors were companion symptoms, both belonging to a cold stage, both associated with a depressed state of the circulation—a state of anæmia, and not a state of hyperæmia. And this view of the matter derives some additional support from the fact that in all cases of neuralgia the patient is apt to shiver and shudder during the paroxysm. There is, in fact, abundant evidence to show that pain of a neuralgic character is associated with a state of circulation which is altogether opposed to the state of inflammation and fever: at any rate there will be no lack of such evidence when what has just been said is taken in connection with what has to be said in the next paragraph and in the paragraphs following.

¶ 135. *Pain of a neuralgic character would seem to be antagonized rather than favored by an overactive condition of the circulation.*

In rheumatic fever the rule, I believe, will be found to be this—that the pains which had been torturing the patient for days or weeks or months previously, preventing him from being comfortable when up, and causing him to toss about in sleepless misery at night, come to an end when the feverish reaction and local inflammation of the fully formed disorder make their appearance. After this, the joints are *tender* enough, but if the patient keep as still as he is very likely to do under the circumstances, he is comparatively at peace so far as pain

is concerned. Or if it be otherwise, the pain will generally be found to be in a part in which the signs of rheumatic inflammation are imperfectly established or absent, or else at a time in which there is a decided remission in the feverish symptoms—an event which happens more frequently in this disorder than is commonly supposed.

It is also difficult to look upon the local inflammation of gout as essential to the existence of the racking pain of this disorder. "About two o'clock in the morning," says Sydenham, who knew full well from personal experience what to say, "the patient is awakened by a severe pain in the great toe, or, more rarely, in the heel, ankle, or instep. This pain is like that of a dislocation, and yet the parts feel as if cold water were being poured over them. Then follow chills and shiverings, and a little fever. The pain, which was at first moderate, becomes more intense; and with its intensity, the chills and shivers increase." After tossing about in agony for four or five hours, often till near daybreak, the patient suddenly finds relief, and falls asleep. Before falling to sleep, the only visible change in the tortured joint is some fullness in the veins; on waking in the morning, this part has become swollen, shining, red, tender in the extreme, and more or less painful, but this painfulness is as nothing when compared with the torture of the night past. It seems, indeed, as if the pain which now exists must be referred to the mere tension and stretching of the inflamed ligaments, for it may be relieved, or even removed, by judiciously applying support to the toe and to the sole

of the foot. On the night following, and not unfrequently for the next three or four nights, the sharp pain in all probability returns, reappearing and disappearing suddenly or almost suddenly, and resulting in the discovery of additional inflammatory swelling upon awaking in the morning. The pain in these relapses, like the primary pain, is accompanied by chills and shivers, and by the most distressing irritability and excitability, but until unequivocal signs of inflammation are developed in it the painful part is not tender in the full sense of the word. The inflammation is attended by no fever, or by very little; or if it be otherwise, as it is occasionally, the inflammation runs higher than usual, *and the characteristic pain is less urgent than usual.* Dr. Garrod points out this latter fact in his excellent work on Gout,* and says that he has seen several illustrations of it. From its history, then, it would seem as if the inflammation of gout were not essential to the pain of gout. It would seem as if the pain went hand in hand with the rigors which are preliminary to the development of the inflammation. It would seem as if the inflammation had little to do with the pain, for, if it were otherwise, it is scarcely to be supposed that the pain should be least urgent in the cases of gout in which the inflammation is most marked, and that the unequivocal signs of inflammation should make their appearance during sleep without waking the patient. Nay, it would even seem as if the pain were put an end to by the establishment of the inflammation—

* "Gout and Rheumatic Gout." Post 8vo. London: Walton and Maberly, 1859. p. 39.

as if, in fact, the pain were antagonized rather than favored by the inflammatory condition. Moreover, the suddenness with which it begins and ends in the majority of cases must be looked upon as a reason for referring the pain to the category of neuralgia—a category of which, to say the least, inflammation cannot be regarded as an essential characteristic.

There is also reason to believe that pain holds the same relation to fever and inflammation in other kinds of fever besides the rheumatic and in other kinds of inflammation besides the gouty.

A few days ago I saw a patient in the Westminster Hospital who complained of violent pains all over the body, especially in the back and loins, and also of chills and shivers. A few hours afterward he was hot and feverish, and the pains and chills and shivers had all taken their departure. The case was one of small-pox; and the lesson it conveyed to my mind was that the pains and the rigors were symptoms which ought to be classed together, and considered as belonging to the cold and not to the hot stage of the fever. And this case would seem to be a fair illustration of what happens in other fevers; for it seems to be the rule rather than the exception for the pains which attend upon the onset of these disorders to pass away or to become greatly mitigated when the cold stage gives place to the hot stage. Nay, it would even seem as if pain gave place for the time to what may be called artificial feverishness. At any rate, I have more than once felt tic douloureux pass away as soon as I could set my blood fairly in motion by violent

bodily exercise; and on two occasions I have derived a similar benefit from a practice which is not unfrequently adopted in the hunting field, and put an end summarily to a sudden attack of lumbago by leaning forward in the saddle and beating the loins with the two hands until the whole body was aglow and the perspiration dropped from the forehead.

Nor is it different with inflammation. In the case of a dislocation or sprain, for example, the acute pain of the accident—the pain to which Sydenham likens that of gout—does not, as a rule, remain after the parts have begun to be hot and swollen and tender; and this case is certainly no exception in the history of inflammation. It would seem, in fact, as if the proper place for the pain was among the phenomena of the preliminary cold stage—the stage of "shock," and not among the phenomena of actual inflammation. And it is not impossible that the efficacy of blisters in the relief of many kinds of pain may furnish another passage in a similar story; for it is a fact which is as well established as any fact in therapeutics, that blisters are most effectual means of relieving pain, and that this relief is usually coincident with the blistering—that is, with the inflammation set up by these agents. Nor is a contrary conclusion to be drawn from the history of certain cases in which pain continues as a permanent symptom after the full establishment of inflammation, as, for example, in deep-seated inflammation of the mamma in the female, in orchitis, and in inflammation of the hip-joint; for in these cases it is a fact that this persistent pain

is immediately relieved or removed by those operative measures which diminish the tension or stretching arising directly or indirectly from the inflammation. It is a fact, that is to say, that the persistent pain in these cases is an accidental and not an essential accompaniment of the inflammation—a consequence, as I have just said, of tension or stretching of the tender tissues, and not a necessary part of the inflammation itself.

How far these inferences will be confirmed or set aside by the histories of those forms of pain in which the nervous system is more especially implicated remains to be seen; but so far there seems to be good reason for believing that pain of a neuralgic character is connected with a depressed state of the circulation rather than with the opposite state of febrile or inflammatory excitement.

¶ 136. *Pain the result of tenderness, and not pain of a neuralgic character, would seem to be associated with the state of active congestion or inflammation.*

This is a necessary corollary to what was said in the last paragraph; and, as will be seen presently, what was there said is only a part of what might have been said with reference to this matter.

(2) On the pathology of pain as deduced from the condition of respiration during pain.

¶ 137. *The condition of the respiration during pain sheds no very certain light upon the pathology of pain.*

One or two facts might be cited here which tend to show that deficient respiration is favorable to the

production of pain; but these facts are of somewhat uncertain significance, and, therefore, I will not waste the little time at my disposal in dwelling upon them. One thing, however, is plain, and this is, that the respiration cannot be overactive during pain of a neuralgic character if the circulation at this time be in the condition which has just been described.

(3) On the pathology of pain as deduced from the condition of innervation during pain.

¶ 138. *There is reason to believe that pain of a neuralgic character is antagonized rather than favored by inflammatory excitement of the nervous system.*

About thirteen or fourteen years ago, I had an opportunity of becoming acquainted in my own person with the history of facial neuralgia, or tic douloureux. At that time I was anything but strong and well, and the pain, so long as it lasted, did not tend to improve my condition, for it took away my appetite, and kept me awake at night. I was dejected also, and troubled with frequent chills and shivers. For the first two or three days after the commencement of an attack the painful part of the cheek would admit of pressure, and the face was pale and perspiring. On the third or fourth day the painful cheek became swollen, hot, and tender, a state of general feverish reaction was developed, and contemporaneously with these changes the true neuralgic pain came to an end. I had abundant opportunity for knowing that this was the true order of these changes: first, neuralgia without

local tenderness and swelling and redness, and with frequent chills and shivers, and a decidedly depressed condition of the circulation; and, afterward, local tenderness, redness and swelling, with general feverish reaction, without chills and shivers, and without neuralgia. I also find that my own experience in this matter is the exact counterpart of the experience of several patients who have come under my notice at different times.

It is also the rule rather than the exception for toothache to come to an end when the face becomes swollen and inflamed; and it does not seem to be otherwise with the stabbing pains which so generally precede the inflammatory eruption of herpes, for these pains scarcely ever remain after this eruption is fully developed. Nay, I can call to mind three cases of sciatica in which the relief of the neuralgic pain was coincident with the development of tenderness at one or more points in the course of the painful nerve, and in which, after this change, the patient was comparatively free from pain so long as the lame limb was kept still and let alone.

With respect to neuralgia in all its manifold forms one thing is certain, and this is, that neuritis is not necessary to its production. All are agreed upon this point; but all are not agreed upon the next point—namely this, that severe pain is not a necessary accompaniment of cerebral or spinal meningitis, or of neuritis.

Pain certainly is no very conspicuous symptom in the common form of cerebral meningitis—that is, in the tubercular form; and in simple meningitis there is reason to believe that any severe pain in the head

is the precursor of, rather than the attendant upon, the actual inflammation. Two days ago I saw a well-marked case of acute simple meningitis in a boy aged fifteen. When I first saw him, my patient complained of agonizing pain in the head, with frequent chills and shivers; and at this time his face was pale and perspiring, his ears and his head generally somewhat below the natural temperature, his pupils somewhat dilated, and his pulse contracted and feeble. Eight hours afterward, when I saw him a second time, his face was flushed, his head burning hot, his pupils contracted, his eyes ferrety, his skin hot and dry, his pulse strong and excited, *and fierce delirium had taken the place of the pain.* And this, so far as my own experience goes, is the regular course of this disorder. It is pain ceasing, not beginning, as the symptoms of active determination of blood to the brain make their appearance. It is pain in association with an anæmic rather than with a hyperæmic condition of this organ.

About five years ago I had in the Westminster Hospital a case of a young man, aged 23, in which after death there were symptoms of recent spinal meningitis of an acute character. The illness began three days before admission by sharp pain in the back and legs, shiverings and retention of urine, these symptoms making their *débût* within a short time after sleeping flat on the back in a meadow. Upon examination the back was found stiff, with the head drawn back considerably, and very severe pain was experienced along the whole course of the spine, in the legs, in the lower part of the abdomen, and less severe pain in the head. The pain in the back

was somewhat aggravated by pressure, and by the application of a sponge wrung out of hot or cold water. The patient lost power rapidly, and died at the end of a week; there being very little febrile excitement of the circulation during the time that he was under observation in the hospital, and but little impairment of the power of moving the limbs. The common sensibility of the whole surface was increased, but not to any marked degree. The bouts of contraction and pain were very occasional and of very short duration during the last three days of life; and it was certain in many instances that they would have been avoided if the patient could have kept perfectly quiet. It was probable, in fact, that the pain, like the muscular contraction, was the result of movement—the result of tenderness. It was probable, that is to say, that the pain was the result of tenderness, and not pain of a neuralgic character. The history of this case, indeed, agrees very fully with the history of the cases already given when speaking of the relation of spasm to acute spinal meningitis (¶ 129); and both these histories must, I think, corroborate the conclusion already drawn—that pain of a neuralgic character is antagonized rather than favored by inflammatory excitement of the nervous system. And certainly this is the conclusion which must be drawn from the history of those painful spinal disorders so often met with in hysterical patients, for here pain of a severe neuralgic character is a prominent symptom, and yet the collateral symptoms and the issue of the disorder in nineteen cases out of twenty make it impossible to ascribe this pain to inflammation in the cord or its membranes.

I cannot appeal to my own experience for a well-marked case of neuritis, but I may perhaps be permitted to refer to the three cases of sciatica already mentioned for an illustration which is better than none. In these cases, the extreme local tenderness, with some degree of swelling, seemed to show that neuritis was developed in the course of the sciatica, and yet there was at this time no increase of pain. On the contrary, the plain fact was rather this—that the severe neuralgic pain, which had existed for some time previously, was at an end when the swelling and tenderness gave evidence of the establishment of inflammation in the course of the sciatic nerve. I believe, also, that this will be found to be the history of neuritis when this history is recorded by observers who take care to discriminate between pain of a neuralgic character and pain the result of tenderness.

In cases, then, in which the nervous system is especially implicated there is little reason to believe that pain of a neuralgic character is a sign of inflammatory excitement in this system. On the contrary, the general tenor of the evidence would seem to show that such pain points to a condition of the nervous system which is altogether opposed to such excitement.

¶ 139. *Pain the result of tenderness, and not pain of a neuralgic character, would seem to be associated with a state of inflammatory excitement in the nervous system.*

Some direct evidence in support of this proposition is mixed up with the evidence which was advanced

in the last paragraph in support of the proposition that pain of a neuralgic character is antagonized rather than favored by inflammatory excitement of the nervous system. Such evidence, for example, may be found in the history of the cases of neuralgia in which it is to be presumed that neuritis is developed in the course of the disorder—cases in which the nerve changes from a state of comparative indifference to pressure into a state of exquisite tenderness and in which at the same time the previous torture comes to an end if only the nerve be let alone. Some collateral evidence in support of the same proposition is also to be found in the section in which it was attempted to deduce the pathology of pain from the condition of the circulation in this disorder, for it is to be supposed that the tenderness developed in the course of any local inflammation is the result of the *nerves* of the part having been made to participate in the inflammatory change. It would seem, in fact, that pain is only an accidental feature in inflammation, for pain the result of tenderness is only the sign of exalted sensibility—a phenomenon to be explained in the same way as the sensation produced by the prick of a pin or by any other means. It is not present if the inflamed part be let alone. It is not essential like pain of a neuralgic character: it is only accidental.

¶ 140. *There is reason to believe that pain of a neuralgic character is associated with a state of irritation in the nervous system and not with the state of inflammation.*

It has been seen that pain of a neuralgic character

is often associated with a state of circulation which is the very opposite of that state of excitement which is met with in active fever and inflammation: and in the cases in which pain seems to be associated with active fever and inflammation it has been seen that the place of the pain is in the cold stage before the establishment of the hot stage of the disorder, and not in the hot stage itself—in the stage of irritation preliminary to the inflammation, and not in the stage of actual inflammation. And I have nothing new to say upon this point at the present time.

¶ 141. *There is reason to believe that inflammatory excitement of the nervous system is a consequence and not a cause of the state of which neuralgic pain is the sign.*

The history of true neuralgic pain, as already given, is a proof that this kind of pain is anterior to the establishment of the inflammation, and, therefore, it may be supposed that this inflammation stands in the relation of a consequence rather than in that of a cause to the pain. Moreover, the connection of pain of a neuralgic character with a state of irritation in the nervous system is calculated to show that inflammation in the nervous system must be looked upon as the consequence rather than as the cause of the pain; for it has been shown, on more than one occasion already, that the state of irritation (which implies a state of contraction in the vessels) may give rise to inflammation when it is carried far enough to exhaust and paralyze the vasomotor nerves. All, in fact, that has been urged to show that inflammation is the consequence of that

state of irritation which leads to convulsion, tremor, or spasm, may be urged to show that inflammation may be the consequence of that state of irritation which leads to pain of a neuralgic character.

¶ 142. *There is reason to believe that pain of a neuralgic character is to be regarded as a sign of defective vital power in general, and of defective nerve-power in particular, and not of a contrary state of things, and that, in this respect, pain is the exact equivalent of convulsion, tremor, and spasm.*

This, as it seems to me, is the broad conclusion which is fully borne out by the evidence advanced in the present and in the preceding lectures; and what is required now is, not to enforce it by new arguments, but only to call attention to it.

¶ 143. *There is reason to believe that the view of the pathology of pain which is here taken is in perfect accordance with the theory of sensation which has been propounded in these lectures, and also that these two views reciprocally interpret and corroborate each other.*

When speaking upon the physiology of sensation, I came to the conclusion that there is no essential difference between the action which issues in sensation and the action which issues in muscular contraction. I showed that in the case of a sentient nerve, as in the case of a motor nerve, the nerve loses electricity when it passes from the state of rest into that of action. I showed that the change in

a sensory nerve when sensation is produced by the action of voltaic electricity, and the change in a motor nerve when muscular contraction is produced by the same means, are exact equivalents. Taken by itself, perhaps, the evidence was not altogether conclusive; taken in connection with what had been previously said respecting the physiology of muscular motion, it acquired a meaning which was scarcely to be misunderstood. In a word, the conclusions respecting the physiology of sensation were such as to make it probable that some important changes would have to be made in the current views respecting the pathology of pain—changes such as those which have just been indicated—changes identical with those which were found to be necessary in the pathology of convulsion, tremor, and spasm. And thus it is that this view of the pathology of pain is in perfect accordance with the physiological view of sensation which has been taken previously, and that each view supplies to the other the particular support of which it stands in need when taken by itself alone.

§ II. ON THE THERAPEUTICS OF PAIN.

The few remarks which have to be made under this head have reference only to the treatment of neuralgia and nervous pain. Indeed, with respect to the treatment of the pain resulting from tenderness all I can find time to say is this—that the pressure upon which it depends must be avoided or obviated,—that in many cases rest is the grand remedy,—and that in some cases, probably in

many, it may be supposed that the local hyperæmic condition upon which the pain depends may be intended to repair a damage done in a previous state of irritation, and that instead of combating it, all that may be necessary is to let it alone,—to leave nature free to work in her own way.

¶ 144. *The avoidance of damp and cold appears to be of paramount importance in the treatment of neuralgia and nervous pain.*

The investigations of Ahrens, Nasse, and others, of which I gave an account in my first lecture (¶ 5), show that the natural electroscopic evidences of animal electricity become very obscure or altogether wanting in the rheumatic condition, and, doing this, they afford some reason for believing that the electrical relations of the exterior and interior of the nerves at the seat of pain are reversed in the way in which they are found to be reversed when nerves are in a state of irritation (¶ 47), for it has been seen that this reversal is apt to happen when the natural electricity of the nerves becomes very feeble. Hence it is not difficult to see that cold may be favorable to the production of pain by preventing the development of electricity in the nerves of sensation, for this development of electricity appears to be in direct relation to the activity of the circulation, and that damp may greatly aid in bringing about the same result by favoring the conduction of electricity away from the body. And if so, then it follows as a natural consequence that warmth and dryness may be very beneficial in the treatment of neuralgia and nervous pain, and that

flannel, cotton-wool, silk, resinous plasters, and other non-conductors, applied to a painful part, may do all the good which is ascribed to them by common consent.

¶ 145. *The diet best suited for the neuralgic habit of body appears to be one which does not contain too much lean meat and too little fatty and oily matter.*

Looking at this matter theoretically, and assuming the correctness of the previous physiological and pathological conclusions, there is the same necessity for the adoption of this rule of diet in the case under consideration as that which existed in the case where convulsion, or tremor, or spasm, was the disorder to be prevented or cured. Nor does this matter appear in a different light when regarded from an empirical point of view. I have often met with persons of a nervous and neuralgic habit who have sedulously avoided butter and fatty matter for fear that these articles would give rise to biliousness, and who have lived almost entirely on lean meat and strong soups, in the vain expectation that this kind of nourishment was that which was necessary to restore or supply the strength which was obviously wanted; and I do not remember a single instance in which relief of pain, disappearance of the headache and sickness designated "biliousness," and improvement in general health, did not rapidly follow the addition of oleaginous articles of food to the diet, and the due adjustment of fibrinous articles. I have carried out this idea in practice for four or five years, and I have now no doubt that the cases in which this rule of diet is that which ought

to be adopted form the rule and not the exception. Indeed, I have lately had the idea that the training for the ring, in which lean butcher's meat figures so conspicuously, may have the effect of nourishing muscle too much and nerve too little, and that this may be one reason why pugilists have (as it seems to me) so often broken down in their powers of endurance, and why the process of training itself can never be kept up beyond a certain limited time with advantage.

¶ 146. *There is reason to believe that sugar in excess may be harmful in a neuralgic habit of body.*

I have now seen several cases of obstinate rheumatism in which the patients were very fond of sugar, and I think I may say, without hesitation, that in all these cases the diminution in the allowance of sugar had the effect of mitigating the pains. I have, also, been led to think that the sugar may have done harm in these cases by favoring the formation of that substance which, upon very good grounds, is believed to have so much to do with the production of rheumatic fever—viz., lactic acid; and I have not yet found any reason for regarding this view as a fallacy.

¶ 147. *The properly regulated use of alcoholic drinks would appear to be an essential part of the preventive and curative treatment of neuralgic pain.*

As in the case of convulsion, tremor, and spasm, so in the case of pain of a neuralgic character, what the system seems to want is to be roused to a higher degree of vital power. What the system wants in

these cases is precisely that which the proper use of alcoholic drinks promises to supply. Theory points to this conclusion, and practice does not contradict it. On the contrary, I have no hesitation in saying that the proper use of alcoholic stimulants is at once the natural corrective of the neuralgic habit and the most trustworthy of all anodynes. I have repeatedly known a paroxysm of neuralgia prevented and cut short by a glass of hot grog, and the condition of the patient in other respects improved rather than damaged by the proceeding; and this is more than I could say of any other method of treatment. And I have too often seen the beneficial influence of rum and milk in the morning in correcting the neuralgic habit to have any room left for doubt upon this score. In a word I am satisfied, on practical as well as on theoretical grounds, that the proper use of alcoholic drinks is an essential part of the preventive and curative treatment of nervous pain and neuralgia, strictly so called.

¶ 148. *There is reason to believe that coffee or chocolate, or cocoa, is to be preferred to tea as a common beverage in the management of neuralgic cases.*

Coffee, chocolate, cocoa, and tea are usually supposed to act in the same manner, because their alkaloidal principles—theine, caffeine, and theobromine —are identical in chemical composition, and, no doubt, their action is somewhat similar. They all, under ordinary circumstances, favor wakefulness. They all serve to correct the excitement or stupor arising from the too free use of alcoholic drinks. But there are differences as well as analogies in the

action of these beverages, and, practically, I find it to be as I have said, that coffee or chocolate is to be preferred to tea as a beverage in neuralgic cases. Theoretically, I cannot readily explain why it should be so. Coffee, perhaps by virtue of the empyreumatic substances which are generated during the process of roasting, may have some special restorative powers. It may do good as a stimulant. Chocolate may be suitable on account of its oily nature, doing good in the same way as that in which all oleaginous matters do good in neuralgic cases. Cocoa may do some good, because the hot water in which it is infused is a stimulant, and because it has no positive power of doing harm. Tea, on the other hand, after the stimulating effects of the hot water in which it is infused have passed off, must, under ordinary circumstances, be regarded as a sedative. In fact, cold infusion of tea must be regarded as not remotely analogous in its action to digitalis, and, therefore, it may be supposed that tea may only be tolerated as a beverage in cases where there is a more active condition of the circulation than that which is met with in the neuralgic habit of body. But be the reason what it may, I am perfectly satisfied that it is of importance to eschew tea as an habitual beverage in any case where there is a marked disposition to nervous pain or neuralgia, and that the same objection does not apply to coffee, or chocolate, or cocoa.

¶ 149. *There is reason to believe that the habitual use of purgatives and aperients is a pernicious practice in persons of a neuralgic habit.*

I have come to the conclusion that aperients will scarcely ever be wanted if a sufficient amount of oleaginous matter be introduced into the diet, and that, as a rule, a patient is less liable to pains of a neuralgic character, when he has got into the state in which these oleaginous matters are made to do the work which is so often assigned to purgatives and aperients. I am, indeed, very much inclined to believe that there is no one practice which so much tends to keep up the tendency to nervous pain and neuralgia, as that of using aperients and purgatives habitually. And for the rest I will only say that the remarks which I made on the subject of aperients and purgatives in another lecture (¶ 105) are equally applicable in the present case.

¶ 150. *There is reason to believe that cod-liver oil may be a very suitable tonic in many cases of neuralgia and nervous pain.*

I have now for some time been in the habit of giving cod-liver oil in many cases of neuralgia and nervous pain, with, as I believe, marked benefit. In some cases, no doubt, there were special features in these cases which suggested the use of quinine, or iron, or iodide of iron, in place of the oil, or along with it; but in the majority of these cases the oil was tried by itself, or along with some preparation of phosphorus, and with the result which has just been stated. It seemed to me that cod-liver oil was indicated in these cases for the same reasons as those which made it expedient to increase the oleaginous articles of the food; and it now seems to me that these theoretical reasons have not been contradicted by the results of practice.

¶ 151. *There is reason to believe that phosphorus may be indicated in many cases of a neuralgic character.*

In the cases of neuralgia or nervous pain in which I have given cod-liver oil and the analogous articles of food, I have generally given some preparation of phosphorus, as the phosphorated oil, or the ethereal tincture.* I have given the oil and the phosphorus for the same reason—as elements required for the proper nutrition of nerve-tissue. I have given them because the pathology of pain obliges me to believe that the energy of the nervous system is deficient in neuralgic cases, and that this energy may be repaired by improving the nutrition of the nerve-tissue. I have, indeed, applied to the treatment of pain the same principle as that which I applied to the treatment of convulsion, tremor, or spasm; and, so far, I may say without hesitation that I have found no reason to be dissatisfied with the result. I hope before long to illustrate this point by the narration of cases; and in the mean time all I can do is to make this general statement.

In speaking in this manner of phosphorus as a remedy in neuralgic conditions, however, I do not intend to regard this substance as a specific in all cases, or in any case at all times. I do not intend to say that other remedies, as quinine, iron, arsenic, iodide of iron, iodide of potassium, bromide of

* Since the delivery of this lecture I have given the hypophosphites of soda or magnesia, or lime, as substitutes for the galenical preparations mentioned in the text, and the reasons for, and the results of, this change are the same as those which are stated in the footnote on p. 207.

potassium, and others, are not to be preferred to it, or associated with it, in certain cases—in many cases, perhaps, at certain times: on the contrary, I fully recognize the fact that in neuralgia, as in all other morbid conditions, it is the patient which must be treated rather than the disease, and that the success of the treatment will always depend upon the tact with which the peculiarities of a particular patient are recognized and provided for.

¶ 152. *There is reason to believe that electricity in certain forms may be useful in the treatment of neuralgia and nervous pain.*

The conclusions at which I have arrived respecting the electrical condition of the system have led me to believe that it may be necessary to return to the original mode of using electricity as a therapeutical agent. They have led me to believe that good would arise from insulating the patient and charging him with positive electricity. To do this is to do what promises to improve the natural electrical condition of the system, for this condition is one in which the exterior of the nerve and muscular fibers is electrified positively. To do this, also, is to do what promises to correct that state of irritation in nerve-fiber which is the essential cause of pain, convulsion, tremor or spasm, for in this state of irritation, the electrical relations of the exterior and interior of the nerve-fibers are reversed at the seat of irritation, so that the exterior of the fibers is negative instead of positive. Acting upon this idea I have placed several persons suffering from neuralgia and nervous pain upon an

insulating stool, and kept them charged with positive electricity for some time: and I think I may say that the almost invariable result was relief of pain at the time and for some time afterward. In some of these cases the patient was kept charged without taking sparks from him: in other cases a succession of sparks was taken from the painful part. In these latter cases the operation was often followed by an erythematous rash, and by decided and not very transitory relief to the pain: and I am disposed to think that this counter-irritant use of electricity will prove to be of great importance as a means of relieving pain. As yet, however, my experience upon this point is not of sufficient extent to allow me to refer to it with any degree of confidence.

I am also disposed to think that a succession of shocks from an induction-coil will have a very beneficial influence in this treatment of some forms of neuralgia and nervous pain, *provided only the operation be carried on long enough to bring about vascular reaction—long enough to bring on an artificial hot stage by paralyzing to a certain extent the vaso-motor nerves.* Used in this way, I am disposed to think that the shocks might be a means of urging the system out of the stage of irritation, which is coincident with the pain, into the hot stage after the cold stage—the stage of reaction, which is antagonistic to pain rather than favorable to it, and I have already seen three cases which seem to give not a little countenance to this notion.

¶ 153. *There is reason to believe that counter-irritants in certain forms may be made of much use in the treatment of certain kinds of pain.*

If inflammation be antagonistic to the state of irritation which produces pain rather than favorable to it, it is easy to understand how it is that blisters, actual cautery, and other modes of counter-irritation, are, what they are most undoubtedly, such effectual means of relieving pain. And this is all that need be said upon this subject.

¶ 154. *There is reason to believe that sedatives in sedative doses are not often required in the treatment of neuralgic or nervous pain.*

At times, without doubt, morphia and its congeners in the materia medica may be necessary in the treatment of neuralgia and nervous pain, but not, I believe, in doses which destroy the power of feeling pain. What I believe is that these medicines must be given as tonics or stimulants, rather than as sedatives, in order to produce a satisfactory result. As with alcoholic drinks, it is necessary to give them so as to sustain or rouse the vital power, and not to stupefy it, to favor natural sleep by removing the irritation which prevents it, and not to bring on in its stead the stupor of drunkenness, so, I believe, it ought to be, and so it may be, with the remedies which are called sedatives or narcotics because they are generally administered only in stupefying and intoxicating doses. In fact, I hold that no remedies are necessarily stimulating, tonic, or sedative—that in a given dose, and in a given state of the system,

any remedy may be made to act as a stimulant, or tonic, or sedative; and with respect to morphia and its congeners I believe that these remedies will not act beneficially in the relief of pain of a neuralgic character if they be given in sedative, that is in stupefying doses—that to do this is to cut the Gordian knot, and not to untie it.

VII. ON PARALYSIS IN CERTAIN ASPECTS.

The few moments remaining at my disposal do not permit me to be otherwise than very brief in what I have to say under this head.

§ I. ON CERTAIN QUESTIONS RELATING TO THE PATHOLOGY OF PARALYSIS.

¶ 155. *The fact that paralysis is accompanied by morbid muscular contraction, as twitchings, cramps, convulsions, and so on, is no certain proof that the paralyzing lesion is of a congestive or inflammatory character, but rather an argument to the contrary.*

All that has been said in these lectures upon the physiology and pathology of muscular motion is confirmatory of this statement; and I know of nothing in the history of paralysis which is in contradiction, except the mere belief that morbid muscular movements are signs of exalted functional activity in some part of the nervous system, and that there *must* be hyperæmia in this part to account for this exaltation.

¶ 156. *The fact that paralysis is accompanied by pain of a neuralgic character is no certain proof that the paralyzing lesion is of a congestive or inflammatory character, but rather an argument to the contrary.*

In the remarks which have just been made upon the pathology of pain it has been shown that pain of a neuralgic character is associated with a state of anæmia rather than with a state of hyperæmia; and so far as I know there is nothing in the clinical history of paralysis to militate against this conclusion.

¶ 157. *The fact that paralysis is accompanied by morbid sensations, such as formication, itching, pricking, coldness, heat, weight, tightness, and so on, is no certain proof that the paralyzing lesion is of a congestive or inflammatory character.*

The natural inference from the premises is that morbid sensations of any kind denote a state of irritation and not a state of inflammation in some part of the nervous system. It is very possible, however, that some of these sensations arise from undue tenderness, and that this state of undue tenderness is the result of congestive or inflammatory change; and, therefore, I do not venture to dogmatize upon the pathological significance of these sensations.

¶ 158. *The fact that paralysis is not accompanied by morbid muscular contraction and by pain and other morbid sensations is no certain proof that the*

paralyzing lesion is not of a congestive or inflammatory character.

This fact, according to the premises, is rather to be looked upon as an argument to the contrary; and, so far as I know, this inference is not contradicted by any passages in the history of paralysis which have not been mentioned.

¶ 159. "*Late rigidity*," *or that kind of rigidity into which paralyzed muscles pass eventually, and which is a permanent and not a transitory phenomenon, would appear to have some close analogies to rigor mortis.*

In his writings on paralysis, the late Dr. Todd spoke of "early rigidity" and "late rigidity" as accompaniments of paralysis. "Early rigidity" is evidently a form of spasm. As its name implies, it happens at an early stage of the disorder, and, like spasm, it has to be referred to some "state of irritation" in some part of the nervous system. It only happens now and then, and it may pass off. "Late rigidity" is quite different. It is the invariable consequence of uncured paralysis if the patient live long enough. It comes on equally whether "early rigidity" have been present or absent; and it never ends in relaxation. It does not commence until the paralyzed muscles have wasted considerably; it is established by slow degrees, and, when it is perfect, the contracted and wasted muscles have altogether ceased to respond to the action of galvanism. Dr. Todd accounts for this form of contraction in this manner: "At the seat of the original lesion, whether

it is simply a white softening or an apoplectic clot, or a red softening, with more or less destruction of brain-substance, there takes place an attempt at cicatrization more or less perfect. Attendant on this, there is a gradual shrinking or contraction of the cerebral matter, which, acting on the neighboring healthy tissue, keeps up a slow and lingering irritation, which is propagated to the muscles, and excites in them a corresponding gradual contraction, while at the same time their nutrition becomes seriously impaired by the want of proper exercise, and by the general depressing influence of the lesion."* Thus wrote Dr. Todd. As it seems to me, however, a much more easy explanation of "late rigidity" is that which is supplied by the view of muscular motion set forth in these lectures—namely this, that the muscles have lost their innate electricity and vitality when the rigidity comes on, and that they contract and remain contracted in consequence of this loss—a view in which the aid of "irritation" is altogether dispensed with, for according to it, "late rigidity" is the anticipation of rigor mortis—*rigor mortis in vitâ*, and nothing less or more.

¶ 160. *An increased disposition to reflex movement is no proof that the paralyzing lesion is of a congestive or inflammatory character, but rather an argument to the contrary.*

The cases of paraplegia in which the legs exhibit a marked disposition to reflex movements, so far as my experience goes, are precisely those cases in

* "Clinical Lectures on Paralysis, and other Diseases of the Nervous System." Post 8vo. Churchill.

which the most careful examination fails to find in any part of the spine any of the tenderness which may be supposed to indicate a congestive or inflammatory condition of the subjacent cord. I have notes of three remarkable cases which illustrate this point in the clearest manner; and I have also notes of several cases of paraplegia, most of them of no long standing, in which decided tenderness in some part of the spinal cord was associated with diminution or actual annihilation of all reflex movements in the legs. Judging from what I myself have observed, it would seem, not only that a congestive or inflammatory condition of the cord was *not* necessary to the production of increased reflex movements, but that such a condition had a positive influence in counteracting such movements. As in the case of the frog whose spinal cord has been cut across, so in these cases, the essential condition in the production of increased reflex movements would seem to be the isolation of the parts in which the movements are manifested from the great cranial nervous centers by the damage done to the cord as a conductor, and nothing more.

¶ 161. *The fact that paralysis is absent in some cases in which the conductors of nervous influence are disorganized in a certain part of their course is not altogether unintelligible.*

I have lately had in the Westminster Hospital two very remarkable cases in which a part of the spinal cord was diffluent (as like as possible to ordinary cream), and yet the state of paralysis and loss of sensibility were by no means so great as might have

been expected in the parts below the lesion; and cases of this kind are, I believe, by no means uncommon. How, then, is this? Is the explanation to be found in the experiments of Humboldt which were cited in the first lecture (¶ 1)—experiments which show that nervous influence may act across an actual gap in the nerve-texture? This is a question which seems to demand an answer in the affirmative rather than in the negative; for if nervous influence can act as it is seen to do in these experiments, it is not difficult to believe that it may also act in a case where the place of an actual gap is filled up with altered or disorganized nerve-tissue.

§ II. ON THE THERAPEUTICS OF PARALYSIS.

According to the premises, the fact that paralysis is or is not accompanied by morbid muscular contractions or morbid sensations has a very different bearing upon the treatment of the paralyzing lesion to that which it is usually supposed to have. According to the premises, indeed, the signs which are generally regarded as indicative of a congestive or inflammatory character in this lesion must be read in a contrary sense, and *vice versâ*. Hence, for example, it may be necessary to give strychnia in a very different class of cases to those in which it is commonly given. Hence many other changes which must be obvious, and upon which I must leave my hearers to form their own inferences. Indeed, all I can now do is to say a hurried word upon two points concerning which I desire to express an opinion.

¶ 162. *There is reason to believe that the proper use of electricity will be of much service in the treatment of many forms of paralysis.*

The remarks which have been made in the present lecture upon the use of electricity in the treatment of neuralgia and nervous pain are applicable here; and the sum of the whole matter is that there is reason to hope that a new field in the therapeutics of paralysis will be opened out—by charging the body with statical positive electricity,—by drawing sparks in the course of the disabled nerves,—or by using the shocks of the coil-machine until the operation is followed by a sufficient degree of vascular reaction. In chronic cases, I think it may be possible, by the latter means, to improve the nutrition in the faulty nerve-center by producing a state of vascular reaction there, for it may be supposed that the shocks of the machine will affect the nerve acted upon throughout its course, and that forthis reason the vaso-motor nerves of the vessels of the nerve-center will not be out of reach of this action. In a word, I have already seen several very satisfactory results from employing electricity in one or other of the three ways which have been mentioned; and I am not a little sanguine as to the good to be done by persevering with this practice.

¶ 163. *There is reason to believe that the proper use of movements and manipulations will be of service in the treatment of many forms of paralysis.*

Orthodox medicine has much to learn from heterodox medicine in this matter, and it is to be hoped that no time will be lost in recognizing this

fact and in acting upon it. It is to be wished, indeed, that the results obtainable in paralysis and in many other cases by means of the treatment called Lingism (after its originator Ling), or the Swedish system of gymnastics, or kinesitherapy, or the movement-cure, were more generally known and appreciated at their proper value. Surely it is not right to refuse to recognize a truth because it happens to be presented in a manner which is more or less erroneous. Surely it is not right to neglect an important means of cure because many (certainly not all) of those who are alive to its merits, and who carry it out practically, are quacks and impostors, and in alliance with other quacks and impostors. Surely the practitioners in orthodox medicine are electics, ever learning, and ever bound to learn and apply every means of healing. For my own part, I may say that I have long been in the habit of using various movements and manipulations in the treatment of paralysis, and that I am every day more and more convinced that to omit such movements and manipulations in these cases is to deprive the patient of a most important aid to recovery.

APPENDIX.

BEFORE the time when my essay on the "Philosophy of Vital Motion" appeared in public, others had written with a similar object in view, and published what they had written, but I was not then aware of the fact, nor did I become aware of it for four or five years subsequently.

The name which must be mentioned first in order is that of Dr. West, of Alford, in Lincolnshire. As early as 1832,* in some remarks upon the influence of the nerves upon muscular contractility, this writer maintains, "that the nervous influence which is present in relaxed muscular fiber is the only influence which the nerves of volition possess over that tissue; that its office there is to restrain or control the tendency to contract which is inherent in the muscle; and that contraction can only take place when by an act of the will this influence is suspended, the muscle being then left to act according to its own innate properties;" . . . and again, "that nervous influence is imparted to muscular fiber for the purpose of restraining its contraction, and that the action of the will, and of all other disposers to contraction, is simply to withdraw for awhile this influence, so as to allow this peculiar property of muscular fiber to show itself." The coexistence of spasmodic action with nervous debility, the efficacy of stimulants as antispasmodics, and the postponement of rigor mortis until all traces of nervous action have disappeared, are the principal facts which are adduced in support of the probability of this theory.

Very shortly after the publication of these remarks, a similar idea appears to have been hinted at by Sir Charles Bell in a lecture at the Royal College of Surgeons in London, for, after premising that the question could never be settled, the lecturer said, "that *relaxation* might be the act, and not contraction, and that physiologists, in studying the subject, had too much neglected the consideration of the mode by which relaxation is effected." This remark is preserved by Dr. West in the essay to which reference has just been made.

* "On the Influence of the Nerves over Muscular Contractility"—London Medical and Surgical Journal. Edited by Michael Ryan, M.D., vol. i. 1832.

Six years later, in a chapter of his classical work on comparative anatomy,* Professor Dugès, of Montpellier, argues with much clearness that all organic tissues are the seat of two opposite movements—expansion and contraction, and that contraction, which is in no sense peculiar to muscle, is nothing more than the cessation of expansion—"la contraction musculaire ne consiste que dans l'annihilation de l'expansion." The muscle is supposed to contract in virtue of its elasticity, just as a piece of caoutchouc must contract when set free from a previous state of extension; and an analogy is hinted at between the expanded state of the muscle and the fluid state of the fibrine of the blood, and between rigor mortis and the coagulated state of this fibrine. Analogous in its effects to electricity, the vital agent is supposed to accumulate in the muscles, and to produce expansion by causing the muscular molecules to repel each other; and contraction is supposed to be brought about either by the sudden discharge (as in ordinary contraction) or by the gradual dying out (as in rigor mortis) of the vital agent. And, further, it is supposed that the rhythmical movements of muscle are caused by successive discharges of the vital agent, which discharges are brought about whenever this agent acquires a certain degree of tension; and that the cramps of cholera, or the spasms of tetanus or hysteria, are consequent upon the development of the vital agent being for the time suspended.

More recently still, namely in 1847, Professor Matteucci communicated a paper to the Academy of Sciences at Paris† upon the influence of the nervous *fluid* in muscular action, in which he writes:—"Ce fluide développé principalement dans les muscles, s'y répand, et, doué d'une force répulsive entre ses parties, comme le fluide électrique, il tient les éléments de la fibre musculaire dans un état de répulsion analogue à celui présenté par les corps électrisés. Quand ce fluide nerveux cesse d'être libre dans le muscle, les éléments da la fibre musculaire s'attirent entre eux, comme on le voit arriver dans la roideur cadavérique. . . . . Suivant la quantité de ce fluide qui cesse d'être libre dans la muscle, la contraction est plus ou moins forte." Professor Matteucci appears to have framed this hypothesis, partly, in consequence of certain considerations which seemed to show that the phenomenon of "induced contraction" was owing to the *discharge* of electricity in the muscle in which the "inducing contraction" was

* "Traité de Physiologie Comparée de l'Homme et des Animaux." 8vo. Montpellier and Paris, 1838.

† "Comptes Rendus." March 17, 1847.

manifested—an idea originating with M. Becquerel—and, partly, in consequence of the analogy which he himself had found to exist between the law of contraction in muscle and the law of the discharge in electrical fishes; but he does not appear to have attached much importance to the hypothesis. Indeed, his own comment at the time is —"j'ai presque honte d'avoir eu la hardiesse de communiquer à l'Académie des idées si vagues, et apparemment si peu fondées, et contre lesquelles on pourrait faire bien des objections, mais je pense que, parmi les théories physiques les mieux fondées aujourd'hui, il on existe qui ont débutés de cette manière, et il est certain que des hypothèses, aussi peu fondées que celles-ci, ont quelquefois peut produire ensuite des découvertes remarquables."

Next in order, and almost contemporaneously with the date of my own first publication on the subject, Professor Engel, of Vienna, wrote: *—"So hat der Nerve die Aufgabe, nicht die Zusammenziehungen des Muskels zu veranlassen, sondern den Zusammenziehungen bis auf einen geringen Grad entgegenzuwirken. Im lebenden Organismus, in welchem Ruhe etwas unmögliches ist, ist auch ein ruhender Muskel eben so wohl wie ein ruhender Nerv undenkbar, der Muskel in seinem beständigen Streben, sich zusammenzuziehen, wird vom Nerven daran verhindert, im Nerven macht sich das fortwährende Streben kund, die Zusammenziehung des Muskels auf ein gerechtes Mass zurückzuführen; das Ergebniss dieser zwei einander entgegengesetzen Eigenschaften der Nervens und des Muskels ist das, was man gemeinhin Zustand der Ruhe, Zustand des Gleichgewichtes, oder an Muskeln auch Tonicität nennt. Das Verlassen dieses Gleichgewichts ist die Bewegung einerseits, die Lähmung andererseits. Die Bewegung wird aber erzeugt, indem entweder der Einfluss des Nervens auf den Muskel herabgesetz wird, oder indem die Contractionskraft des Muskels unmittelbar gesteigert wird. Lähmung des Muskels findet sich gleichfalls entweder durch unmittelbare Vernichtung der Contractionskraft des Muskels oder durch eine übermässig gesteigerte Einwirkung des motorischen Nervens auf den Muskel. Sollen daher abwechselnde Muskelcontractionen zu Stande kommen, so ist die Gegenwart des lebendigen Nervens im Muskel unerlässlich, und auch bei unmittelbaren Muskelreizen können abwechselnde Zusammenziehungen nur erfolgen, so lange noch die Nerven lebensfähig sind; hört letzeres auf, so zieht sich der Muskel ohne Hinderniss zusammen. Diesen Zustand nennen wir die Todtenstarre." The chief grounds

* "Ueber Muskelreizbarkeit," "Zeitschrift der Kais. Kön. Gesellsch. der Aertze zu Wien," Erster Band, pp 205–219, and pp. 252–270, 1849.

for this opinion are, first, certain original experiments, some of them very remarkable, which afford additional proof that the muscles of frogs are more prone to contract when they are cut off from the influence of the great nervous centers; secondly, the frequent spontaneous occurrence of cramps and other forms of excessive spasmodic contraction in paralyzed parts; and, thirdly, the supervention of the permanent contraction of rigor mortis when all signs of nervous irritability are completely extinguished.

And, last of all, I find Professor Stannius, of Rostock,* arriving at the conclusion—"dass es eine wesentliche Aufgabe der sogenannten motorischen oder Muskelnerven sei, die natürliche Elasticitätsgrosse der Muskelfasern herabzusetzen und ihre Elasticität vollkommener zu machen; dass anscheinende Ruhe des Muskels, zum Beispiele, während des Schlafes, das Stadium solchen regen, den Muskel zu seinen Aufgaben weider befähigenden Nerveneinflusses anzeige; dass active Muskelzusammenziehung einen geregelten und begrenzten momentanen Nachlass des Nerveneinflusses auf den Muskel bezeichne; dass endlich die Nachweisung einer Muskelreizbarkeit, in der üblichen Auffassungsweise, ein durchaus vergebliches Bemühen sei." M. Stannius was led to this conclusion by certain original experiments in which he found blood to have the power of relaxing rigor mortis and restoring muscular irritability, and these experiments are advanced in evidence. Reference is also made to arguments, to be brought forward on another occasion, which will prove—"dass diesse Anschauungsweise, so paradox sie immer auf den ersten Anblick sich anlassen mag, mit unserem thatsächlichen Wissen über Nerven-und Muskelthätigkeit keineswegs im Widerspruch steht." The essay from which these quotations are taken was published toward the end of 1852—about two years after the date of my first publication on the subject.

[From the Introduction to my work, entitled "Epileptic and other Convulsive Affections of the Nervous System; their Pathology and Treatment." Third Edition (incorporating the Gulstonian Lectures). Post 8vo. London: Churchill, 1861.]

* "Untersuchungen über Leistungsfähigkeit der Muskeln und Todtenstarre," Vierordt's "Archiv. für Physiol. Heilkunde." Stuttgart, 1 heft, 1852.

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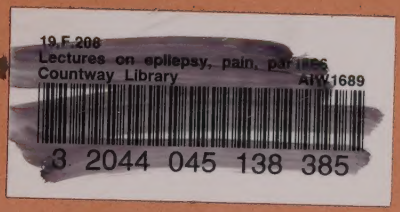
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